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GENERAL SURGERY

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GENERAL SURGERY

A PRESENTATION OF THE SCIENTIFIC PRINCIPLES UPON WHICH THE PRACTICE OF MODERN SURGERY IS BASED

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AMERICAN EDITION

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WITH FOUR HUNDRED AND FORTY-NINE ILLUSTRATIONS IN THE TEXT, PARTLY IN COLOR, AND TWO COLORED PLATES

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PREFACE

My associate, Dr. Lewis, and myself have undertaken the translating and editing of this text-book on General Surgery, written by Prof. Erich Lexer, because we believe that it presents the present status of the subject of general surgery in a more thorough and complete way than any other text-book.

This work is a presentation of the scientific principles upon which the practice of modern surgery is based.

In text-books on surgery in all languages two great divisions of the subject are made, and to these two divisions of the subject various names have been given. In England and America, the terms the Science and Art of Surgery and the Principles and Practice of Surgery have been generally employed. On the Continent, the terms General and Special Surgery are used.

These latter seem to me preferable, and we have therefore retained the term General Surgery in our American edition of Professor Lexer's work, and hope that this may be adopted by the American and English profession. There are certain general principles of pathology and therapeutics and operative technic which apply to all fields of surgery, and when these are thoroughly mastered by the student or practitioner their application to special surgical conditions becomes at once intelligent and easy and scientific. For him who lacks this knowledge the practice of surgery becomes a handicraft.

The subject of General Surgery should be studied by the student before regional or special surgery is taken up. In our own surgical department, the ground covered in this book is studied in the third year in a course extending over a period of six months, three hours each week being devoted to recitations, conferences, and laboratory work. This work, with the addition of a six months' course in surgical anatomy, is made prerequisite to the study of regional and special surgery.

Practitioners who are interested in surgery will find, I believe, great interest and profit in studying this book. The advances in the science of surgery in the last few years have been so rapid and so great that it has been difficult for the surgeon engaged in active practice to keep abreast of the increasing knowledge. As an example one might men-

tion the significance and importance of the modern conception of infection and immunity, and the application of this knowledge to surgery. Professor Lexer has presented these modern views in a clear, concise, and practical way. This English translation will offer to those who do not read German a most complete presentation of the present status of the Science of Surgery, a department in which our German colleagues excel. Dr. Lewis and I have not hesitated to make such additions and changes as seemed to us desirable to make the book more complete.

Special attention is called to the excellent chapter on Blastomycosis, written by Dr. Oliver Ormsby. This disease has been especially studied in America. The Continental authorities have not had much experience with the disease, and have been rather skeptical about it. I hope that Dr. Ormsby's chapter will be included in the next German edition.

The chapters by Dr. Rosenow, on blood examinations in surgery, and also on the subject of opsonins and the Wright vaccination treatment, are a distinct addition. Dr. George Crile has kindly allowed us to publish an abstract of his recent work on the direct transfusion of blood.

We have retained most of the illustrations of the German edition, and have added a number of plates taken largely from our own clinic.

I desire to express my appreciation of the work of D. Appleton & Company, the publishers, who kindly undertook this publication at my suggestion, and who have spared no pains to make the work acceptable in every way. I feel that Professor Lexer's book is most valuable and timely, and in offering this English edition to the profession I desire to express the hope that it will be widely read, and that those who read it may find it as profitable and instructive as I have.

ARTHUR DEAN BEVAN.

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PART I

I. WOUNDS, THEIR TREATMENT AND REPAIR

CHAPTER I

WOUNDS

Definition.—A wound is a solution of continuity of the external coverings of the body, its mucous membranes, or the surfaces of organs. Wounds are classified as simple if the deeper structures are not injured, as complicated if they are (viz., associated injury of muscles, nerves, large vessels, bones, body cavities, and joints; cf. Part IV).

Different Kinds of Wounds and Causes.—The form of the wound depends upon the character of the vulnerating force, whether sharp, blunt, cutting, crushing, or lacerating. The principal forms of wounds are the incised, contused, and lacerated.

Incised wounds produced by a sharp instrument or object (fragment of glass) have well-defined smooth edges and surfaces, the tissues of which are not otherwise injured. The edges of the wound may, however, be bluish in color, bruised and infiltrated with blood, and the adjacent area swollen, if the wound has been made by an imperfectly sharpened instrument or dull object, or if the blow has been delivered with considerable force, or tangentially, or if a gash has been made with a sharp weapon. The extent to which a wound gapes depends upon its relation to the tension planes of the skin. If the wound crosses these at right angles, for example, if it is transverse on the extremities, longitudinal on the sides of the thorax and abdomen, its edges will be widely separated. The edges of even a large wound may be closely approximated if its direction corresponds to the course of the elastic fiber bundles of the skin. In operative incisions this fact should always be borne in mind, as the resulting scar will be better if the incision follows tension planes (Kocher). (Fig. 1.)

In incised wounds and gashes in which the skin is divided obliquely instead of vertically, flaps are formed which are connected with the body by pedicles of different widths. If the pedicle is entirely cut

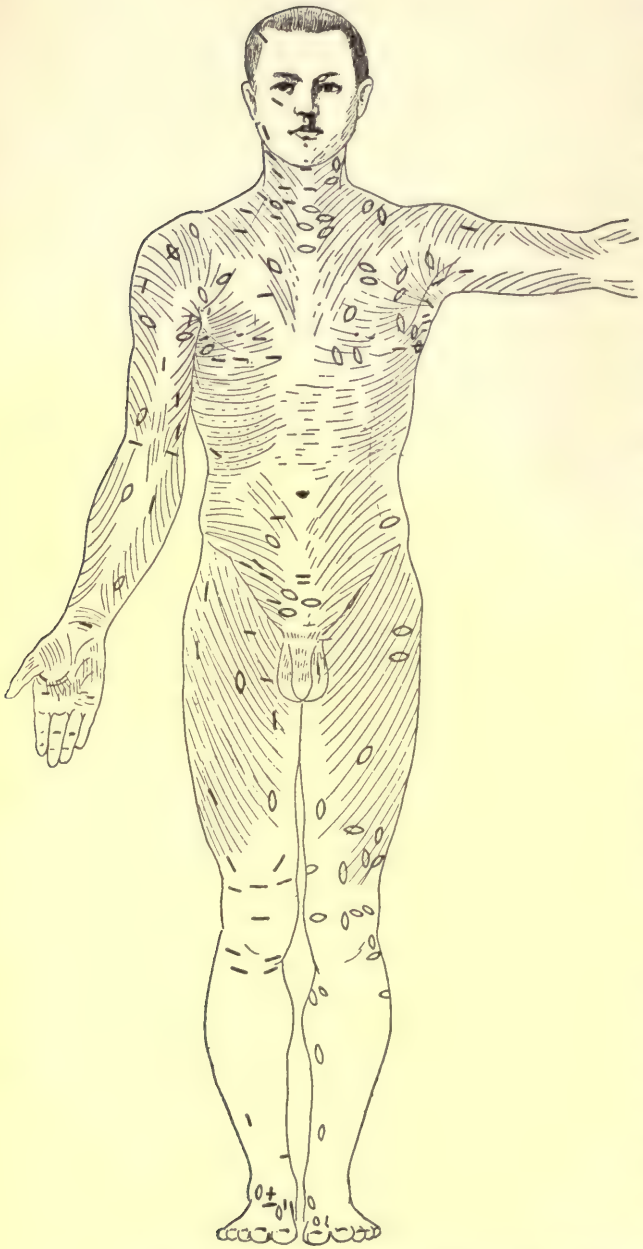


FIG. 1.—DIRECTIONS OF THE TENSION PLANES OF THE SKIN.

across there is then a loss of substance or a defect. Such wounds are produced by blows which are delivered almost parallel to the surface of the body.

Punctured wounds have the characteristics of incised wounds. They are always narrow and deep, corresponding somewhat in diameter to the penetrating weapon or instrument (viz., needle, nail, dagger, sword, lance, arrow, trocar). They gape little because they are so narrow that their edges often adhere. Their edges are contused and lacerated only when the wound is produced by a blunt object, such as a picket, a cane, or an umbrella.

The borders of contused wounds are irregular, discolored, and raised by the exudation of blood into the tissues. Abrasions of the skin, subcutaneous hæmorrhages, ischæmia, and loss of sensibility indicate the extent of the cutaneous area involved in the injury

produced by the blunt force. If in addition the subcutaneous tissue is crushed and separated from the skin by an oblique thrust or blow, the edges of the wound may be raised from the subjacent tissues and large pockets filled with blood coagulum formed.

Contused wounds are more frequent where bones lie near the surface than where a large amount of soft tissue intervenes. The gaping of contused wounds depends not only upon their direction, but also upon the amount of cutaneous and subcutaneous blood infiltration. In subcutaneous injuries of the abdomen contusions of the stomach and intestines and of those viscera which may be forced against the vertebral column are relatively common.

Lacerated wounds are produced by blunt objects, the force being applied obliquely, with a resulting tearing and rupture of the skin or mucous membrane. Their edges are irregularly torn, but the surrounding area is less involved than in contused wounds. Bursting wounds and rupture of organs resemble lacerated wounds. Such wounds of the skin are produced by explosions, as, for example, lacerations of the cheeks caused by the discharge of a weapon into the mouth with suicidal intent. Lacerations of vascular organs or those filled with fluid contents (spleen, liver, kidney, heart, bladder, stomach, intestine) are produced by the application of blunt force to their surfaces, or by projectiles penetrating with great velocity.

Many wounds are produced by the simultaneous contusion and laceration of tissues, and are then frequently accompanied by the formation of flaps or the loss of substance. Machine injuries, bites, scratches, and gunshot wounds belong to this class. In gunshot wounds, the grooved or grazing wound in which the projectile passes parallel to the skin is differentiated from the penetrating wound, which has a wound of entrance, and, if the projectile perforates, a wound of exit. Contusions and lacerations are common in large wounds of exit, in those cases in which the projectile strikes transversely, and in injuries produced by fragments of a bomb.

Immediate Symptoms.—The immediate symptoms caused by a simple wound are local and consist of pain and hæmorrhage. General symptoms (shock, anæmia) more frequently accompany complicated wounds and those associated with profuse hæmorrhage. The pain at the time of the injury and following it varies in intensity, depending upon the susceptibility of the patient, the cause of the injury, and the part of the body involved. The more rapid the separation of tissues, the sharper the penetrating object, the less the pain is. Wounds of the lips, tongue, tips of the fingers, external genitalia, and anal region are especially painful. The pain in the wound caused by the exposure of sensory nerve fibers is perceived as a burning or throbbing sensation.

Pain disappears after the application of a dressing or rest of the wounded part; most quickly in clean, rapidly agglutinating wounds and sutured incised wounds (operation-wounds). After gunshot wounds and severe contusions, the wound and adjacent area may be completely insensitive for some hours or days (local wound-stupor, tissue-shock). This condition, due to the concussion of peripheral nerves, is frequently accompanied by symptoms of mild shock (paleness, cold sweat, apathy, and unrest); (cf. Shock, Part IV).

Hæmorrhage.—Hæmorrhage in a simple wound is greatest if the separation of tissue is produced by a sharp instrument. The troublesome hæmorrhage following a superficial razor cut is well known to everyone. The greater the contusion and the laceration, the less the hæmorrhage, for the contused and lacerated vessels do not remain open so long as when cut transversely. They are closed by agglutination in contusions and by the inversion of their walls in lacerations; also by the coagulation of the blood discharged into the tissues. This difference in closure is found even in large vessels. Punctured and gunshot wounds with fine, narrow channels rarely bleed, as they are occluded by blood clots and closed by muscle tension.

Classification.—Hæmorrhage may be classified as capillary or parenchymatous, venous and arterial. In capillary hæmorrhage the blood oozes steadily from the wound surfaces. Separate bleeding points which may be seen after sponging and become rapidly lost in the general oozing, correspond to small vessels. Arterial hæmorrhage is recognized by the bright red blood, which is discharged in spurts which increase with the pulse beat. Only in dyspnœa is dark blood discharged from the arteries. Venous hæmorrhage is recognized by the dark blood which is discharged in feeble spurts from the large veins. Venous hæmorrhage is greatest when there is an obstruction to the return venous flow, resulting in stasis. The most severe venous hæmorrhage, aside from that due to injury of large veins, follows injuries of venous plexuses (pampiniform, pterygoid, corpora cavernosa penis).

Primary hæmorrhage, which follows immediately the reception of a wound, is differentiated from secondary hæmorrhage, which may follow after some days (six to ten) the mechanical separation or destruction by suppuration of the thrombus occluding the vessel.

The prognosis of repair is most favorable in incised wounds, for here there are no recesses or niches to harbor bacteria and no contused or detached particles to become necrotic and disturb healing. Wounds with contused and lacerated edges and surfaces afford the most unfavorable prognosis. Inflammation easily arises, and the separation of necrotic fragments leads to protracted healing with the formation of

granulation tissue. The most important requirement in operation-wounds is that they be clean-cut, and that contusion and laceration of the tissues be avoided.

CHAPTER II

THE TREATMENT OF WOUNDS

CONTROL OF HÆMORRHAGE

THE first indication in the treatment of wounds concerns the hæmorrhage, the control of which may be temporary and permanent.

Temporary Control of Hæmorrhage.—The temporary control of hæmorrhage in accidents is the duty of the first aid. He who performs this, whether layman or doctor, should know that he does more harm than good if he infects the wound. This consideration may be neglected only in hæmorrhages from large vessels which threaten life. Compression of the wound with the bare hand, handkerchiefs, sponges, and other articles favors and increases the dangers of infection. The popular method of irrigation with hæmostatic agents (cold spring water, ice water, vinegar, alum, salt solution, zinc chloride) by which bacteria are carried from the surrounding area into the wound or from its surface into its depths, and the resulting eschar formation favor the development of infection. For these reasons the irrigation and the tamponing of wounds are to be avoided, and the control of the hæmorrhage by direct compression to be permitted only when sterile dressings are at hand. Without wiping away the dirt and coagulated blood, gauze is laid upon the wound, care being taken that the part of the gauze touched by the fingers does not come in contact with the wound surfaces.

Control of Hæmorrhage by Pressure.—In wounds of the extremities a few turns of a roller bandage applied over the gauze from the periphery afford sufficient pressure to control the hæmorrhage from small arteries. In case of necessity freshly laundered cloths may be used instead of sterile gauze. If this substitute is wanting, it is better to leave the wound alone than to cover it with soiled dressings.

In severe arterial hæmorrhage digital compression must be applied immediately to the injured artery proximal to the wound. After the division of the small arteries of the extremities (digitales or dorsalis pedis) or subcutaneous veins, elevation to the vertical position generally suffices to control hæmorrhage. In the constriction of the extremity by Esmarch's elastic bandage, which has found practical application and extensive use in the constrictor named after him, we have a method

never to be neglected in controlling hæmorrhage. Several turns of the constrictor are applied to the elevated limb close to the trunk, and then it is noted whether the hæmorrhage ceases or increases, whether the skin is white or cyanotic. If the constrictor produces a venous stasis it should be removed immediately and reapplied more tightly. Any kind of a rope or strap, cloth, or strip of linen may be used in place of the elastic constrictor, and if its ends are tied about a cane or an umbrella, torsion can be made until the constriction controls the hæmorrhage. The simple and easily procured Esmarch constrictor has supplanted the old and unreliable tourniquet and similar devices by which a pad or other resistant object was buckled or bound over the trunk of the artery to compress it against bone.

Fig. 2.—Digital Pressure on Femoral.

Control of Hæmorrhage from Mucous Membranes.—In severe hæmorrhage from mucous membranes (nose, gums, tongue) rinsing with cold water or vinegar, or preferably with a five per cent solution of hydrogen peroxide frequently suffices in case there is no constitutional condition like hæmophilia.

Fig. 3.—Digital Compression of the Brachial Against the Bone.

The Use of Esmarch's Elastic Constrictor.—In operations upon the extremities a temporary control of hæmorrhage by artificial ischæmia,

introduced by Esmarch in 1873, is of great advantage, as the blood is excluded from the part and an excellent view of the bloodless tissue can be had.

Constriction of an extremity, according to Esmarch, consists in the application of a thin India-rubber bandage from the periphery to the trunk to force out the blood. This procedure may, however, be omitted, for the elevation of the extremity for some minutes (preferably during the process of sterilization) accomplishes the same purpose. The application of this bandage from the periphery to the trunk may be dangerous in inflammation and thrombosis, as harmful materials may be forced into the circulation by it. This seems, however, to be more a theoretical danger than an actual one, and should not prevent the use of the bandage when it is greatly needed. India rubber, silk or cotton webbing, provided with a clasp or hooks and eyes, can be used for purposes of constriction. For the more massive portions of the limbs (thigh, shoulder) rubber tubing of a finger's thickness provided with an apparatus for fastening can be used. The band or tubing is applied under tension near the trunk, several turns are made, care being taken that the skin does not intervene between the separate turns of the constrictor. Constriction of the forearm and leg is best accomplished with the rubber webbing or a thin pure rubber bandage (Martin's bandage). A thin rubber tubing suffices for the fingers, toes, penis, and scrotum.

FIG. 4.—ELASTIC BANDAGE.

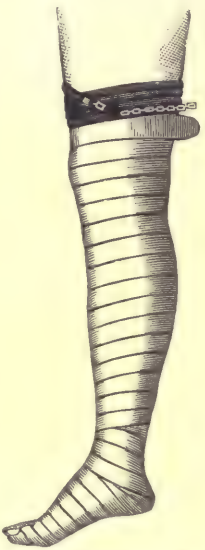


FIG. 5.—ELASTIC BANDAGE APPLIED.

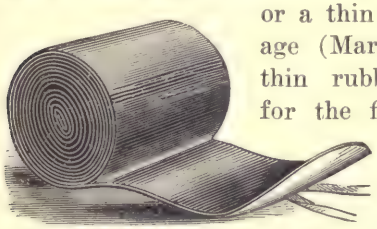


FIG. 6.—MARTIN'S BANDAGE.

During the operation or at its completion (viz., amputation, joint resection, sequestrotomy, incision of phlegmon), all vessels which are visible in the bloodless tissue should be seized and ligated. After procedures in which the wound is not sutured, but tam-

poned because of inflammation, the ligation of the larger vessels suffices, if a well-padded and firm bandage is applied before the constrictor is removed, and the extremity then suspended or elevated for the succeeding twenty-four hours. When the circulation is reëstablished considerable hæmorrhage may occur as the result of a temporary paralysis of the vessel wall due to the constriction. For this reason Esmarch's method has been condemned by some in spite of the fact that such hæmorrhage can be easily controlled. If even pressure with a large gauze compress is made while the constrictor is being removed, and is continued for some minutes while the extremity is elevated, the capillary hæmorrhage (following removal of the constrictor) can be controlled. If the compress is removed the small spurting vessels and bleeding points can be seized and ligated. When the wound is dry it can be sutured.

If the constriction is applied longer than two and a half or three hours, or the rubber tubing applied to weak extremities with considerable force, nerve injuries, ischæmic muscle paralysis, and necrosis of the skin may follow. These results are seen most often in patients who have been transported some distance after their injury or after operation, when the constrictor has not been removed. Esmarch's ischæmia is to be avoided unless very necessary in lymphangitis, thrombosis, and thrombo-phlebitis, because of the danger of separation of the thrombus.

The permanent control of hæmorrhage is obtained by the use of aseptic tampons, compresses, and ligatures. Other methods, such as eschar formation by actual cautery and hæmostatic agents, and angiotripsy, are to be recommended for certain cases only.

Control of Capillary Hæmorrhage and Hæmorrhage from Venous Plexuses.—In accidental wounds, one may proceed to the control of the hæmorrhage after sterilization of the adjacent area. If necessary the Esmarch constrictor may be applied and the wound covered temporarily with sterile gauze while the field of injury is being sterilized. After the removal of the constrictor the bleeding points are ligated as in operation-wounds. *Capillary* hæmorrhage and hæmorrhage from venous plexuses (not controlled by ligature) are easily controlled by a tampon of iodoform gauze, which also stops hæmorrhage by the gradual absorption of fluids by the gauze and resulting swelling, without injuring the tissues or delaying *repair*. This gauze is therefore the most important agent in controlling hæmorrhage from cavities, from the mucous membranes of the nose, vagina, and rectum, and in parenchymatous hæmorrhage following the rupture of vascular organs (liver, spleen, and kidney). The actual cautery has likewise a hæmostatic action in these cases, but is not equal to the *gauze*, because it is more apt to interfere with wound repair. Transfixion suture is frequently of great value in controlling hæmorrhage from vascular organs, kidney, etc.

The Actual Cautery as a Hæmostatic Agent.—The actual cautery, in the form of a platinum point maintained at red heat by benzine vapor, has replaced the hot iron (ferrum candens) which was used in olden times to control hæmorrhage during operations (amputation with red-hot knife). The cautery is valuable in controlling hæmorrhage from mucous membranes, where the tampon cannot be applied—viz., from buccal cavity, hæmorrhoids, and from superficial vascular tumors (hæmangiomas, sarcomas, carcinomas). Where a superficial action only is necessary, the cautery may be replaced by Hollaender's hot-air appa-

Fig. 7.—Paquelin's Thermo-Cautery.

ratus, by which hot air is blown upon the bleeding surfaces. Cauterization of bleeding wounds is to be avoided unless indicated, as the eschar retards healing and favors inflammation. The cautery is being employed less and less each year as a hæmostatic agent.

The same may be said of other hæmostatic agents, such as compresses saturated with liquor ferrisesquichlorati, which forms with the coagulated blood and cauterized wound surfaces a firm eschar which prevents the discharge of wound secretion.

Control of Hæmorrhage in Operation-Wounds.—In operation-wounds any hæmorrhage which is not capillary or parenchymatous is immediately controlled by artery forceps and by ligation. This may be effected in different ways.

Artery clamps (Frick, von Langenbeck, von Bergmann) or artery forceps (Koeberle, Pean, Kocher, etc.) are used for this purpose. They are differently constructed. The extremity which grasps the tissue is ribbed or provided with interlocking teeth. The scissorlike handle is provided with a ratchet lock or clasp.

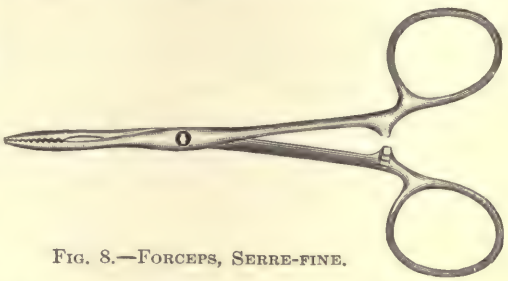


Fig. 8.—Forceps, Serre-fine.

As soon as a vessel is cut it is seized with an artery forceps. The instrument is applied vertically to the wound surfaces, not parallel or obliquely. In this way as little of the surrounding tissue as possible is grasped. If the number of clamps or forceps interferes with the progress of the operation, the vessels can be firmly ligated with catgut and the instruments removed. In case of small vessels the simple is better than the surgical knot; the granny knot should not be used, for it slips easily. It makes little difference what technic is employed in tying the ligature. The fingers should not, however, come in contact with the wound.

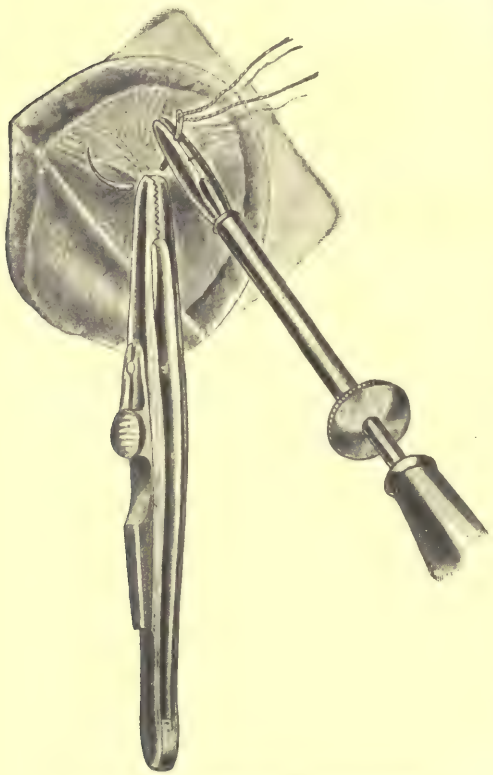


Fig. 9.—Ligation by Transfixion.

In deep parts of the wound artery forceps may be used to tighten the ligatures. Silk may be used in place of catgut in wounds which are not to be closed by sutures. Catgut is generally to be preferred.

Large vessels should be drawn out from the surrounding tissues with the artery forceps and isolated by blunt dissection. A second artery forceps should then be applied transversely, and the first

forcep removed. The ligature then applied to the vessel can be depended upon, for only the vessel and not the surrounding tissue is ligated. Wherever it is necessary to grasp much tissue the artery forceps are not adequate, for in the resistant tissue of the scalp and friable muscle, ligatures do not hold well. They frequently slip and interfere with the progress of the operation or give rise to secondary hæmorrhage. In these cases transfixion is valuable; a needle, carrying a catgut ligature, is passed through the tissue close to the point of the forcep, and tied singly on one side, doubly on the other.

FIG. 10.—TYING LIGATURE AFTER TRANSFIXION.

All visible vessels in loose tissue (subcutaneous, intermuscular, omentum, mesentery, dura) should be seized with two artery forceps before division. The division is then made between the forceps. In this way an operation may be performed with but little loss of blood. Larger vessels are best ligated in continuity, as practiced in courses in operative surgery. The vessel is separated a short distance by blunt dissection from its sheath and bed, and two ligatures are passed around the artery by an aneurysm needle. The ligatures are then tied and the artery cut between them.

In the so-called mass ligature the artery is not directly exposed, but the ligature is carried by a needle or ligature carrier directly through the tissue (viz., omentum, mesentery, peritoneal adhesions) and the structures are ligated in mass.

FIG. 11.—PASSING OF LIGATURE BENEATH ARTERY WITH ANEURYSM NEEDLE.

Control of Hæmorrhage by Torsion of Vessels, Angiotripsy, etc.—Control of hæmorrhage by torsion, by twisting of the applied artery forceps, is too unsafe to supplant ligation. It may be used to the best advantage where small arteries have been seized. Likewise angiotripsy, by which is understood a crushing of a vessel with powerful forceps, is unreliable. This procedure was formerly practiced as forced compression (forcipressure). Hæmorrhage does not occur after removal of the forceps, but

sponging of the wound or muscular action frequently provokes it. In deep, inaccessible wounds (e. g., vaginal operations) long, crushing forceps are used. In order to guard against intermediate hæmorrhage from the uterine arteries they are allowed to remain from twenty-four to forty-eight hours. The control of hæmorrhage by compression of the wound with sterile gauze is made use of as much as possible in every operation. In large wounds the gauze may be pressed against the wound surfaces by the hand, in small wounds by the finger. In skin grafting it is necessary to compress the wound so that the healing of the grafts will not be interfered with by subsequent oozing. Complete dryness of the wound after ligation of all spurting vessels and bleeding points is secured by an even compression of its surfaces, for about ten minutes, with compresses saturated with physiological salt or a solution of three to five per cent hydrogen peroxide. The tissues are not injured by these applications.

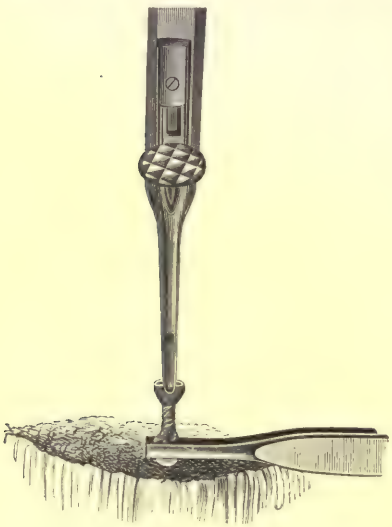


Fig. 12.—Torsion of an Artery.

The Iodoform Gauze Tampon.—The iodoform gauze tampon is indicated after operations in which there are bleeding cavities with resistant walls (sequestrotomies, resection of the maxilla) or wounds with deep sinuses, in which a collection of blood is to be avoided; also after incision of acutely inflamed tissue, for its capillarity not only controls hæmorrhage, but also prevents post-operative absorption (*vide* Treatment of Pyogenic Infections, Part II).

Hæmophilia.—The control of hæmorrhage is most difficult even in the small superficial wounds in patients with diseases of the blood or hæmophilia. By the latter is understood an abnormal condition characterized by a marked predisposition to spontaneous and traumatic hæmorrhages. The essential cause is not known. Lessened coagulability of the blood, abnormal thinness of the vessel walls which are not otherwise changed, and a dilatation of the vessels through vaso-motor influences have all been suggested.

The disease is most frequently congenital, and the male sex is principally afflicted; thirteen times more frequently than the female. Transference from the diseased father, or from the grandfather through a healthy mother, can occur.

Spontaneous hæmorrhages occur especially from the mucous membranes of the nose, mouth, intestine, and bladder, and into the joints, producing often severe changes in the latter (*vide* Hæmarthrosis). Besides, hæmorrhages may occur in the bursæ, the subcutaneous tissue, and kidneys.

The slightest injury of the skin or mucous membrane, such as a needle prick, incised or lacerated wound of the fingers, laceration of the gums in cleaning or extracting teeth, may be followed by severe hæmorrhage, which may continue with slight interruption for days and weeks, and even terminate fatally.

The Control of Hæmorrhage in Hæmophilia.—Often a firm iodoform tampon applied after preliminary cauterization of the wound with the actual cautery will control the hæmorrhage. The pressure by bandages and elevation of the extremity should of course be combined with this treatment. Among the many methods which have been suggested, gelatin has of late received the most thorough trial. According to Dastre and Floresco gelatin increases the coagulability of the blood. It may be used locally in the form of a five or ten per cent solution heated to 104° or 140° F., which is applied to the wound by saturated gauze compresses, or injected into joints. For systemic effects it is injected subcutaneously into the skin of the thorax or abdomen. One to two hundred c.c. of a one to two per cent solution heated to 99° F. are injected daily until the hæmorrhage ceases.

The solution consists of gelatin and physiological (0.9 per cent) salt solution. It is sterilized by heating twice to 248° F., with an interval of one or two days between sterilizations. It is warmed over a water bath each time before using. The use of gelatin is at present a very limited one.

Spontaneous Cessation of Hæmorrhage.—The organism is not defenseless against loss of blood. It is possessed of a number of means of checking and stopping it, which are only successful, however, when the blood is discharged slowly, and not in severe hæmorrhage resulting from the transverse division of large arteries.

It is well known that a simple wound, even an incised wound, ceases to bleed after some time. This is true in operation-wounds, and therefore it is a rule in all operations to grasp bleeding points immediately, not only to limit the amount of blood lost, but also to guard against secondary hæmorrhage from the small vessels, which cease bleeding spontaneously during the operation and would therefore be overlooked.

The change in the size of the lumen of the vessel is the first factor in the spontaneous cessation of hæmorrhage. The lumen is narrowed by the contraction of the circular fibers of the vessel walls; the capillaries are narrowed by the swelling of their endothelium. Vessels, because of

their elasticity, retract from the wound surfaces, and the blood is then forced into the protruding tissues and the vessel sheath. The blood coagulates rapidly and closes the lumen and the lateral wounds of the vessels (*vide* Injuries of Arteries and Veins, Traumatic Aneurysms, etc.).

The spontaneous cessation of hæmorrhage after the transverse division of large vessels depends upon the lowering of blood pressure and changes in cardiac action. A slight loss of blood produces at first a transitory lowering of blood pressure, which is again rapidly restored by a contraction of the vessel walls resulting from an irritation of the vasomotor centers produced by the anæmia. If a large amount of blood is lost, which in animal experiments amounts to more than one fourth of the total quantity, the blood pressure sinks rapidly and the heart beat becomes more feeble.

If the hæmorrhage ceases, the lumen of the vessel is closed by a thrombus since the coagulability of the blood is increased. The lymph, either because the tension of the tissues exceeds the blood pressure or because of the dilatation of the capillaries by vaso-motor influences (Grawitz), flows into the blood, carrying with it numerous leucocytes, and restores the lost fluids.

According to Goltz, death from hæmorrhage results from the empty condition of the heart. The blood pressure becomes so low and the amount of blood is so reduced that no blood is received from the venous system, and after systole the heart does not dilate again.

Dangers of Hæmorrhage Relative to Age, etc.—The dangers of hæmorrhage are greatest in children. The loss of a few c.c. in the new born and of 250 c.c. in a child one year old is dangerous. In the adult a similar danger arises as a rule only after the loss of one half the total amount. Women recover from hæmorrhage more rapidly than men. It is difficult to determine how much of the total quantity of blood (amounting, as a rule, to one thirteenth of the body weight) may be lost without proving fatal, as a number of factors have to be considered.

In the first place the rapidity of loss is a factor, as the danger increases with it. Diseases of the heart and arteries (arterio-sclerosis), anæmia of the brain as in shock, severe anæmias following exhausting diseases, the effects of long operations, and narcosis increase the dangers of hæmorrhage.

AFTER-TREATMENT OF HÆMORRHAGE

Restoration of the Blood.—The blood is restored by absorption of fluids from the tissues; this accounts for the feeling of thirst experienced in hæmorrhage. After a short time the leucocytes (post-hæmorrhagic leucocytosis) increase, after a longer time the red blood cor-

puscles. The time required for complete restoration of the blood depends upon the age, the nutrition, and the condition of the patient. This restoration is to be expected in from two to five days after slight hæmorrhages; in from fourteen to thirty after severe.

Venesection.—Bleeding (venesection), a method current among physicians of an early period, finds no place in surgical practice, which always endeavors to prevent hæmorrhage. In passive congestion, resulting from diseases of the heart or lungs, in chlorosis, uræmia, and eclampsia, bleeding thins the blood temporarily and thus improves the circulation. The composition of the blood is improved by a regeneration of blood cells. The urinary secretion is also increased. Bleeding belongs therefore to the therapeutic measures of internal medicine.

Technic of Bleeding.—Bleeding is performed in the following way: A constrictor is applied to the arm to produce a venous stasis; the radial pulse should not be obliterated. After the field of operation is properly prepared, the skin covering the distended median basilic vein is incised. The vein is exposed at the bend of the elbow and opened for the distance of about 1 cm. The blood is caught in a receptacle and measured; not more than one per cent of the body weight (500–1,000 g.) should be taken. An aseptic dressing is then applied when the bleeding is finished.

The old method of puncturing with the lancet should be discarded, as injuries of nerves and arteries may be produced, and neuralgia and arterio-venous aneurysms result. In every field of surgery incisions should be made layer by layer. Rather make an incision too large and suture it than produce unnecessary injuries.

After some experience the veins can be punctured through the skin with a syringe, as in taking of blood for bacteriological investigation (*vide* Blood Examination in General Pyogenic Infections), and the desired amount of blood removed.

Symptoms of Hæmorrhage and their Treatment.—The principal symptoms of anæmia resulting from hæmorrhage are pallor, pinched features, spots before the eyes, ringing in the ears, weariness, weakness, thirst, rapid, scarcely perceptible pulse, restlessness, anxiety, vomiting, and faintness. Dyspnœa, dilated pupils, loss of consciousness, cold sweat, convulsions, involuntary discharge of urine and fæces indicate the gravest danger. Action must be immediate if this condition is to be successfully treated. At the same time that attempts to control the hæmorrhage are made, agents which strengthen the heart, raise blood pressure, and increase the amount of blood must be used.

The horizontal or, better, partially inverted position of the patient, elevation of the arms and legs and envelopment of the same in an elastic bandage applied with little tension (autotransfusion), wrapping with warm blankets, subcutaneous injection of severel hypodermic syringe-

fuls of camphorated oil, clysters of warm red wine mixed with cloves combat this condition. In patients who are conscious and do not vomit, hot coffee, champagne, mulled wine, hot extract of beef, and other rapidly acting agents may be given, but, most important, normal salt solution slowly and continuously per rectum. If these do not avail, and threatening symptoms are present, salt solution should be given subcutaneously to supply the body with fluids.

Transfusion of Physiological Salt Solution.—The transfusion of physiological salt solution has supplanted the transfusion of blood. The latter is little used to-day, but was practiced in the seventeenth century, particularly by Dieffenbach and Martin. In this method from 140–200 c.c. of blood were removed from a healthy man by bleeding; the blood was then thoroughly defibrinated, filtered through a cloth, warmed over a water bath, and injected into one of the arm veins of the patient. In spite of complete asepsis, chills and fever (so-called transfusion fever, the equivalent of aseptic fever accompanying the absorption of blood exudates) and severe general symptoms (dyspnœa, cyanosis, hæmoglobinuria, bloody diarrhœa, disturbances of consciousness) often followed.

Alterations in the composition of the blood and extensive capillary thrombosis caused these symptoms. Embolism of the vessels of the heart and lungs frequently produced death.

The fatal accidents caused by the transfusion of blood can be explained in two ways. Firstly, aside from the entrance of air into the veins during the injection, small clots, in spite of the filtration of the defibrinated blood, could be injected. Hueter attempted to overcome this by injecting the defibrinated blood into the radial artery, with the idea that the small clots would be retained in the capillaries. Secondly, defibrinated blood contains enough fibrin ferment to make it dangerous because of the possibility of the formation of fibrin.

The direct transfusion [1] of blood from the radial artery of the giver into the arm vein of the receiver has been tried to overcome the disadvantages of fibrin ferment intoxication, a procedure which, however, carries with it the dangers of embolism, for clots readily form about the tube connecting the vessels.

Sheep's blood, employed in earlier times, is even more dangerous than human blood. Its cells and those of the blood of any other species are immediately destroyed and produce extensive coagulation.

The dangers of blood transfusion and the recognition of the fact that the principal cause of death from hæmorrhage is the decrease in blood pressure rather than the alteration of the component parts of the blood have led to the use of physiological salt solution as suggested by

[1] See Appendix.

Kronecker and Sander. The advantages of the salt solution are that its administration is simple, its action immediate, its safety absolute if rightly used.

Preparation of Physiological Salt Solution and Technic of Administration.—Salt solution may be given intravenously or subcutaneously. Where rapid action is necessary and the solution is ready, it is given intravenously. The 0.9 per cent solution can be prepared in any hospital with the boiling water of the steam sterilizer, water being received in sterile pitchers and held in readiness. In practice outside of a hospital, it may be necessary to filter the tap water through gauze and to boil it one half hour in case a sterile solution cannot be obtained from the apothecary. The solution is warmed to 104° F. and poured into a sterile irrigator, to the tubing of which is attached a hollow needle.

After a slight stasis is produced in the arm by the pressure of the hand or a bandage, the canula, the air bubbles having been previously forced out of it, is introduced into the most prominent vein (most frequently the median basilic). If it is feared that the vein will not be found, it may be exposed by a small incision and punctured or opened if a blunt canula is used. In the latter case the vein should be ligated distalward, and a second ligature passed about it proximal to the opening. The solution should be allowed to run into the vein slowly until one or two liters have been given. After the transfusion is completed and the proximal end of the vein is tied and the wound sutured, an aseptic dressing should be applied. In severe cases transfusion may be repeated two or three times in twenty-four hours, and two liters given each time.

The subcutaneous injections are given with large syringes into different parts of the body, most frequently the external surfaces of the thighs, the abdomen, and under the breasts. Too great distension of the skin and too much pressure are to be avoided, because of the severe pain and the danger of necrosis. From one to two liters should be injected, as in the intravenous procedure, and if necessary the injections may be repeated many times.

A very simple and safe method of using the salt solution is to inject it into the rectum, where it is usually readily absorbed, and this is the method to be adopted except in the severe cases where immediate action is imperative.

The success of the transfusion is seen immediately in the improvement of the circulation, and the organism gains time, except in the severest cases, to recover and to gradually restore the quality of the blood. In the severest cases salt solution cannot replace the constituent parts of the blood, and the administration of fluids cannot prevent a fatal issue. In spite of this, transfusion of salt solution has in many cases a life-saving action.

Indication for the Use of Physiological Salt Solution.—Its use is indicated in all hæmorrhages with threatening symptoms without exception, and is often necessary during major operations or at their completion. Accidents resulting from cardiac paralysis and the cardiac weakness accompanying shock may be successfully combated in this way.

The increase of tissue fluids following injections of salt solution improves the general condition when there is deficient absorption of food (e. g., carcinoma cardiæ, vomiting after chloroform, peritonitis). It is used to advantage before operations on poorly nourished patients.

The increased diuresis following the transfusion of salt solution has led to its use in intoxications (e. g., iodoform, carbonic-acid gas, illuminating gas, also uræmia) and in general infections.

Combined Use of Physiological Salt Solution and Oxygen.—In order to better the results of the transfusion of salt solution in severe hæmorrhages, Kuettner has suggested to increase the reduced oxygen content of the blood by the simultaneous administration of oxygen gas. A reservoir is filled with 1,000 c.c. of salt solution, and oxygen gas is allowed to flow in from a tank until 100 c.c. of the solution is displaced. The reservoir is then closed and shaken until the oxygen is absorbed by the solution. Twenty c.c. of oxygen can be introduced with one liter of the solution.

The salt-soda solution (7.5 per cent salt plus 2.5 per cent calcined sodium) recommended by Tavel and used subcutaneously has caused extensive necrosis of the skin.

MECHANICAL, CHEMICAL AND THERMAL INJURIES

Prevention of Infection in Mechanical, Chemical, and Thermic Injuries.—In the care and treatment of a wound there are other important considerations besides the control of hæmorrhage: *The prevention of the entrance of injurious agents, and the restoration of conditions favorable to wound repair.* Wounds should be protected from bacteria, from mechanical, chemical, and thermal injuries.

Operation-wounds should be protected from infection by the rigid observance of an aseptic technic. Every accidental wound should be regarded as infected, for at the time of the injury, more frequently, however, during the period immediately following, bacteria, most often pyogenic and putrefactive varieties, are introduced into the wound. These primary wound infections only become severe, however, if the wound is improperly handled; for example, if an accidental wound, in a condition unfavorable for healing (because of contusion and exudation of blood), is treated as an aseptic operation-wound and closed by sutures. As a rule the secondary infections are much more grave. He

who touches the wound with his fingers, dresses it with soiled gauze, examines its recesses and tract with a probe, other instrument, or finger, washes simultaneously the bleeding wound and its unclean adjacent area, introduces an infection which is of much more significance than the wound itself. It is as much the duty of the first aid to prevent these secondary infections as it is of those who have charge of the after-treatment. In the treatment of the wound the primary infections should not be permitted to develop.

In order to prevent infection during the performance of the first aid a small packet has been devised for use in military practice which consists of a sterile compress and an attached bandage. The arrangement (Perthes, Korteweg) is so simple and ingenious that when opened the gauze comes directly in contact with the wound. These packets, as introduced, for example, by Utermoehlen for emergency dressings into the Dutch army, are of great practical significance, for any wound can be immediately covered with sterile gauze without the danger of infection with unclean hands.

While the clothes of the patient are being removed, the emergency dressing should be allowed to remain, or the wound should be protected by sterile gauze, held in position by a bandage or adhesive strips. The wound should be covered by a dressing while the adjacent area is shaved, washed, and sterilized. If, after the hæmorrhage is controlled and the wound cared for, a dry aseptic dressing is applied, and the examination of the wound with a probe or the finger, even though both are sterilized, and the irrigation or wiping out of its recesses be omitted, the most important thing to prevent *secondary infection* has been done.

There are many ways in which mechanical insults may do harm. Gross mechanical insults naturally do not favor wound repair, as they produce new injuries and cause hæmorrhage. The effects of such injuries produced in treating wounds are rarely seen at the present time, but similar conditions are produced by muscular action, the separation of the edges of the wound, and by the wiping and curetting away of particles of dirt. These insults not only carry the bacteria into the depths of the wound, but also favor their development by injuring the tissues.

Cautious treatment of the wound, and the application of a firm, well-fitting bandage, are most important in the prevention of mechanical injury.

The effects of chemical and thermal influences are seen only when improper or antiquated methods are employed in the treatment of wounds. They injure the tissues, decrease their natural resistance to bacterial invasion, and produce conditions which favor the development and progression of severe inflammations.

The Actual Cautery and Caustics in the Treatment of Wounds.—The effort to destroy completely the cause of inflammation or wound infections led in olden times to the use of a radical measure which we to-day use little for this purpose—the hot iron or Ferrum Candens. All caustics (concentrated carbolic acid, nitric acid, and zinc chloride) have a similar action. The foe is destroyed, but with it the tissue to a great extent, and for this reason cauterization is only occasionally employed in the treatment of wounds. It is used most frequently in those cases in which there is a highly virulent and dangerous infection, for example, in wounds received during the post-mortem examination of fresh cadavers with acute suppurative or general pyogenic infections or anthrax, also in snake bites, hydrophobia, and tetanus. Cauterization of the wound is only reliable if immediate. If the infection is caused by highly virulent bacteria with a short period of incubation, or if time has been allowed for the bacteria to enter the lymph and blood, or if it does not destroy the infectious material, it is dangerous, for the necrotic tissue or eschar closes the wound, and the inflammatory exudate forming behind it cannot find exit, and the inflammation spreads into the tissues.

This is the reason why the knife is better than the cautery, and why severely infected wounds should be excised as quickly as possible after the injury. If a tampon is applied after the wound is excised, the dry gauze takes up and removes the remaining infectious material by its capillarity, and limits the spreading of the inflammation by the removal of the exudate.

Action of Antiseptic Solution.—The weaker the solution of antiseptics (two to three per cent carbolic acid, one half to one per cent bichloride of mercury) used for sterilization, the less the injury to the tissue; likewise, the less the effect upon bacteria. The irrigation of a wound with antiseptic solution never destroys bacteria; it removes mechanically only those lying superficially and attached to blood clots or particles of dirt. Antiseptic solutions never reach bacteria lying within or below the layer of fibrin covering the surfaces of the wound, for the antiseptics form a chemical compound with the albuminous wound secretion by which their action is reduced or destroyed. The bacteria remain, therefore, uninfluenced, while the resistance of the tissues is lowered or destroyed.

Mechanical Removal of Dirt, Hair, etc., from Accidental Wounds.—The cleansing of wound surfaces with chemicals which are injurious should be avoided. The grosser particles of dirt and hair should be removed with forceps, blood clots with sterile gauze, without producing further injury. This can be done by gently irrigating with physiological salt solution, which is allowed to drop from saturated gauze, or more thoroughly with a three or five per cent solution of hydrogen peroxide.

The latter, coming in contact with blood, wound secretion, or pus, liberates oxygen and develops a white foam which, slowly rising from the wound, removes all the superficial dirt with it in the best mechanical way. The solution has the great advantage that it does not injure the tissues, and besides cleansing controls capillary hæmorrhage. According to Honsell the liberated oxygen has no bactericidal action.

The use of the cautery or of antiseptics in the treatment of infected accidental wounds may be compared to the conduct of a campaign by a general, who devastates and damages his own land to undo the enemy. The aseptic treatment of a wound, however (and operation-wounds are placed in this category), attempts to destroy the invading foe by sparing the tissues, and to produce conditions in which he cannot survive.

The Object of the Treatment of Wounds.—The restoration of conditions favorable to wound repair is the object of the treatment of the wound. When one considers all the conditions which favor the development of bacteria in tissues, it is easily understood why the coagulated blood and the necrotic tissue (separated fragments and contused edges of wounds) must be removed, and the wound secretion consisting of blood, lymph, and other exudates drained away. The former is the best culture medium for bacteria; the latter by its accumulation increases the tension of the tissues and drives the infectious materials into the spaces of the surrounding tissues. Therefore the hæmorrhage should be completely controlled and the coagulum removed. Deep recesses should be made accessible by enlarging the wound or making counter openings. Contused and lacerated edges and surfaces of wounds should be trimmed off smoothly with knife and scissors. In old wounds the crust composed of dried wound secretion should always be removed.

The conditions of the wound, together with the possibilities of infection, determine whether it should be sutured or tamponed and drained.

Any incised wound may be sutured provided it has not been improperly treated before being seen by the surgeon, in which case the possibility of infection must be taken into consideration. The clean-cut surfaces of incised wounds do not provide conditions favorable for the retention and growth of bacteria. Some of the bacteria which may have already entered the wound are destroyed by the bactericidal substances in the tissue fluids, while others are removed by the hæmorrhage. In the incised wounds there is no necrosis, and if the control of the hæmorrhage has been complete enough to prevent the formation of a blood clot the conditions are not favorable for the development of bacteria. The same conditions are present in operation-wounds made in tissues which are not infected.

Contused and lacerated wounds should be sutured only in exceptional

cases. If the wound can be rendered clean-cut or excised, it may be sutured completely or partially, when the accumulation of wound secretion is not feared. The lacerated and contused margins of the orifices of the body (lips, nose, eyelids, anus, vagina) should be accurately united, after the edges of the wound have been vivified, to prevent displacement and distortion. Other wounds, if the conditions are not favorable for sutures, should be treated by the open method.

Sutures and Technic of Inserting and Tying.—Interrupted sutures of silk or horsehair are used in closing wounds of the skin. Other suture material, such as silkworm gut, catgut, silver wire, is used but little for skin sutures.

The interrupted suture is the most important, as it can be used anywhere. The method of its application may be seen in the accompanying figures. The sutures as a rule are passed vertically to the edges of the wound; only in exceptional cases, in plastic surgery, where there is a displacement of the skin edges, are they passed obliquely. Sutures used to draw the tissues together under tension are called tension sutures.

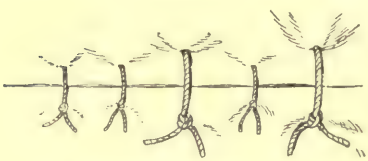


Fig. 13.—Interrupted Suture.

The following technic is employed in the application of the suture. The border of the wound is raised with a toothed forceps, and the needle is pushed through it some millimeters from its edge. When tension sutures are applied the needle should enter the skin about two centimeters from the edges of the wound. The needle is then pushed through the skin until its point appears in the depths of the wound. The other border is then raised and the needle is pushed from the depths of the wound through it. The needle should pierce the skin at the same distance from the edge as on the opposite side.

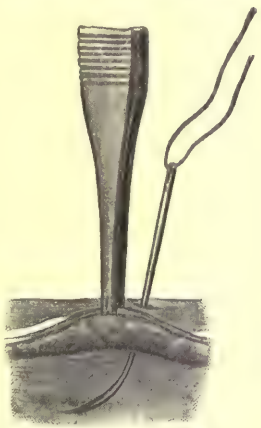


Fig. 14. — Elevation of Edge of Wound with Rat-tooth Forcep While Passing Suture.

If both borders are pierced too superficially, dead spaces filled with blood are formed, which delay wound healing. If the borders are pierced at too great a distance from the edges of the wound and too superficially, the edges will not be approximated, for one will be turned in, the other out (Fig. 15). If in a symmetrical wound one border is pierced deeply, the other superficially, the latter will be turned in and covered by the former (Fig. 16).

In superficial wounds both edges may be pierced at the same time, provided they are held together with tissue forceps by an assistant.

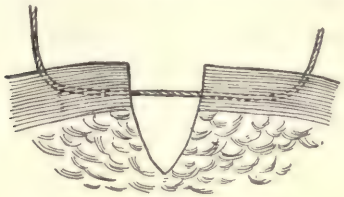


FIG. 15.

FIG. 16.

The surgical knot should be tied. The necessary manipulations may be performed differently, and are a matter of practice. The beginner may practice with thick twine, tying knot after knot, until finally he can do so without paying attention to it.

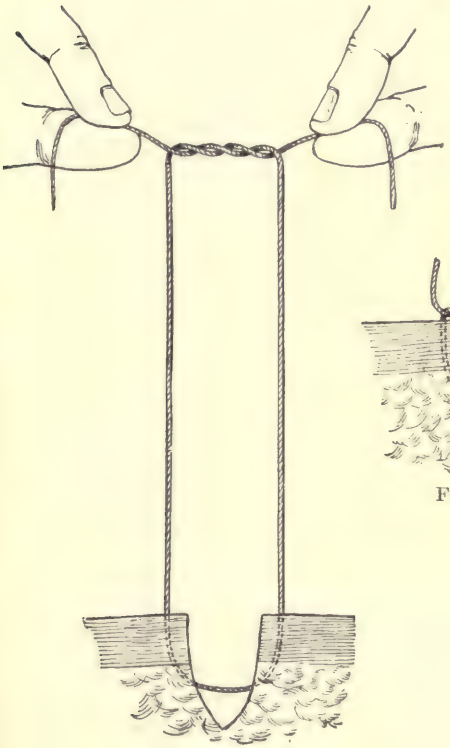


FIG. 17*a*.

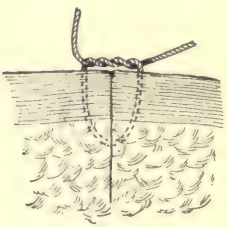


FIG. 18.

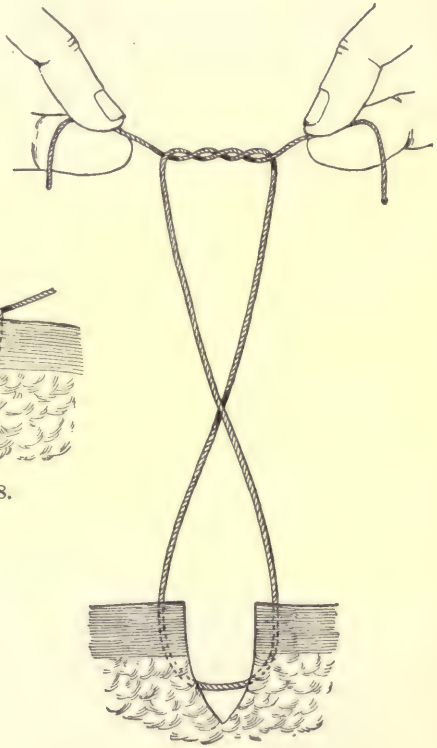


FIG. 17*b*.

In tying a suture both ends should be made tense and held parallel, not crossed, at least a hand's breadth from the wound, as the knot is formed by carrying one end of the suture twice around the other end.

It is impossible to tie the knot quickly if the suture is not made tense, and if tied too close to the wound. This first loop is tightened over the wound until its edges are approximated. Better approximation of the skin edges may be secured if the knot be not tied too tightly. The ends of the suture should then be drawn to the side and the knot displaced, so that it does not rest on the wound; a second simple knot is tied. The ends of the sutures must be changed from one hand to the other while tying the knot, for if they are not a poorly placed "granny knot" will be formed.

If any fat protrudes between the edges of the skin, a superficial suture should be placed at this point. If the edges are turned in, a deep suture should be placed to raise them. If the edges are irregular, the inverted edge should be pierced by a deep suture and the raised edge by

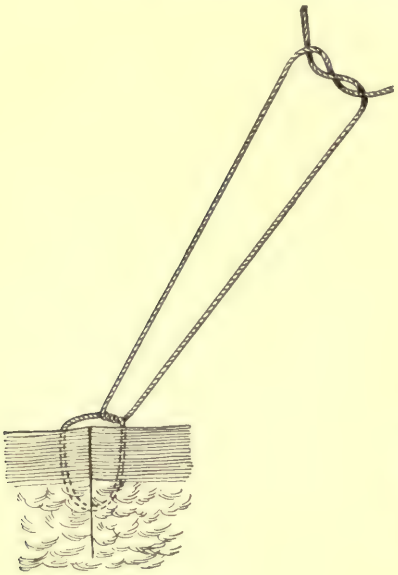


FIG. 19.

FIG. 20.

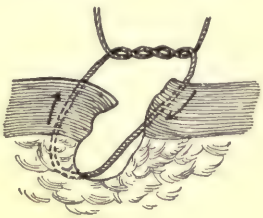


FIG. 21.

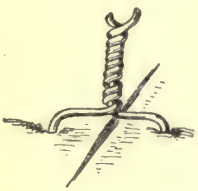


FIG. 22.

a superficial one placed near the margin to equalize the displacement. The same procedure should be used when the edges of a wound are not symmetrical.

The interval between the separate sutures should be on an average about one centimeter. The sutures should be placed at greater intervals when, because of hæmorrhage or contusion, a large amount of wound secretion is feared. In wounds of the face, eyelids, or lips, where a very accurate approximation is demanded, the finest sutures should be used and they should be placed closely together. The advantage of

the interrupted suture is that it is easily applied under different conditions.

If the skin and mucous membrane are cut at the same time, the through and through suture should never be used, for the mucous membrane is usually folded into the wound, and bacteria from its surface pass through the stitch holes. The suture should be passed through the skin to the mucous membrane and out on the other side, not entering on the mucous surfaces, which should be united by a separate row of superficial interrupted sutures.

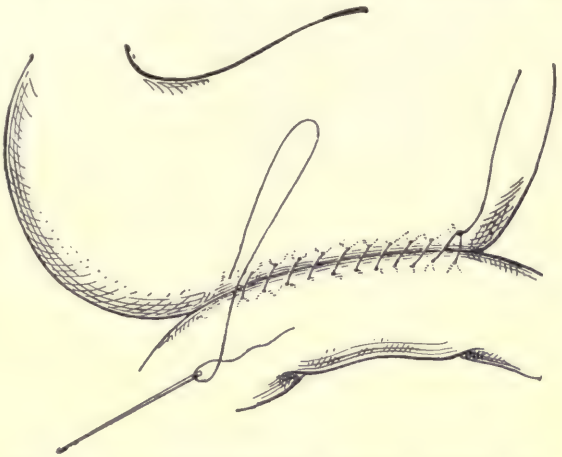


Fig. 23.—Continuous Suture Uniting Stomach and Intestine.

Buried sutures of absorbable catgut, or, where tension is to be prevented, of silk or aluminum bronze should be used in deep wounds, where the anatomical relations of the deeper structures are to be restored or the formation of dead spaces under the skin suture to be avoided. In these cases layer sutures should be used; for example, in closing an incision in the abdominal wall the peritoneum should be sutured first, then the fasciæ and muscles, and finally the skin. Concerning the suture of complicated wounds (nerves, tendons, bones, arteries, etc.) see Injuries of Soft Parts.

Fig. 24.—Mattress Suture.

The continuous suture (glover's or whip stitch) is about the only one of the remaining methods that is used to-day. It is used especially in intestinal work, as it may be quickly applied. After the first stitch is inserted a surgeon's knot is tied; if the suture

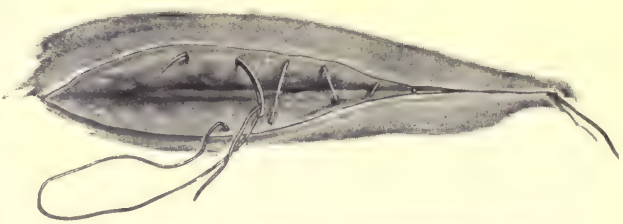


Fig. 25.—Halsted's Subcuticular Stitch.

is not continued to the starting point as in circular intestinal sutures and anastomosis, the stitch is terminated by passing the needle twice

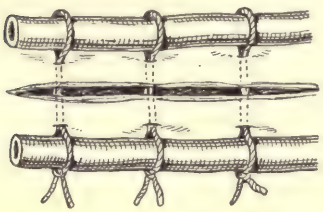


FIG. 26.—QUILLED SUTURE.

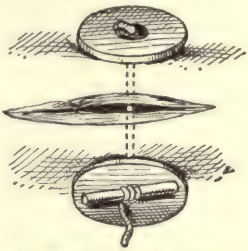


FIG. 27.—LEADED SUTURE.

under the last loop. If so continued the two ends of the suture are tied. At the point where the sutures end and where there is tension, inter-

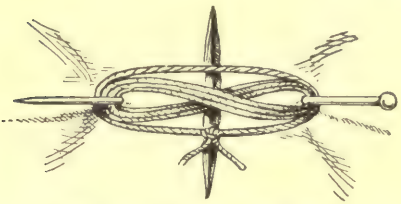


FIG. 28.—TWISTED SUTURE.

rupted sutures may be applied for safety. The mattress, plaited, quilled, and harelip sutures are but rarely used. Halsted's subcuticular suture is more often employed.

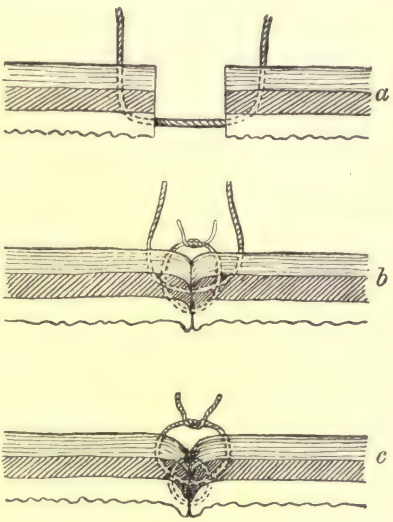


FIG. 29.—INTESTINAL SUTURE. *a*, Inner layer of sutures; *b*, outer layer of sutures (serous layer); the inner sutures are tied; *c*, position of the edges of the wound after tying both layers of sutures.

The Use of Metal Clamps and Fasteners to Close Wounds.—To obviate the cutting of the skin which occurs from the use of sutures different forms of metal clamps or fasteners were introduced by Vidal and others. These were applied to the wound borders with complicated instruments. They can be used only in wounds with symmetrical borders which involve the skin, as they do not grasp the deeper tissues.

Dressing of a Sutured Wound.—The line of suture should be covered with sterile gauze by which the oozing tissue fluids are taken up and dried. The gauze should be held in place by adhesive plaster or collodion, the latter being applied to the edges of the gauze and not directly over the wound. The direct application of collodion pastes or powders

to the wound (iodoform, dermatol) is not to be recommended, for they prevent the discharge of wound secretion, resulting from slight inflammation or suppuration in a stitch hole. Skin sutures should be removed between the fifth and eighth days. If silk sutures are allowed to remain longer they act as a foreign body, and bacteria from the skin invade the tissues surrounding them.

Buried non-absorbable sutures become encapsulated in sterile wounds. If not encapsulated, because of mild infection, a narrow fistula is formed from which the non-absorbable suture of silk or silver wire is discharged, if not previously removed by a dilatation of the fistula.

The fundamental difference between the cutaneous and the intestinal suture is that in the former the separate layers of the wound surfaces are approximated, while in the latter a broad approximation of the serous coats must be obtained by an inversion of the edges of the wound. In intestinal work two rows of sutures should be employed; the first should include all the coats, or the serosa and muscularis, and is hæmostatic; the second the serosa. (Fig. 30.)

Every wound of the stomach or intestine, even if the laceration or contusion does not extend into the lumen, should be sutured, because of the danger of perforation. Fine silk or Pagenstecher's celluloid linen are the suture materials generally employed in intestinal work.

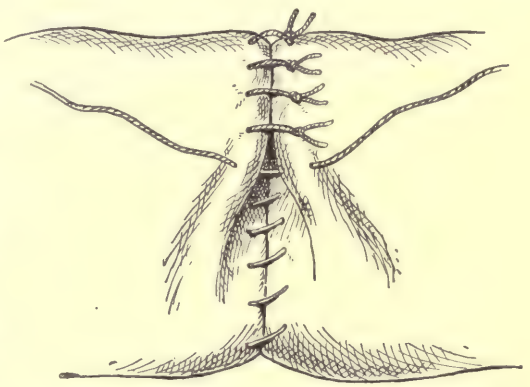


Fig. 30.—The Internal Continuous Suture Is Inverted by an External Interrupted Layer.

Iodoform Gauze Tampon.—If the conditions for wound repair are not favorable, as is frequently the case in contused and lacerated wounds, the open treatment with the the tampon is employed to drain off the infectious material with the wound secretion and to prevent, by allowing free access of air, the development of anærobic bacteria (putrefactive bacteria, tetanus bacilli).

In gunshot and punctured wounds the use of sterile gauze serves a double purpose: (1) It drains away wound secretion, and (2) prevents the development of secondary infections, so that wounds of the deeper structure may heal as subcutaneous wounds without the dangers of inflammation. Iodoform gauze is used as an aseptic tampon. (Concerning its preparation, *vide* Preparation of Aseptic Dressings.)

The iodoform gauze should be laid upon the fresh wound or introduced into wound cavities with sterile instruments and gentle pressure made. After some minutes it becomes firmly attached, controls the hæmorrhage, and removes from the surfaces of the wound by its capillarity (capillary drainage) blood and lymph exudates, and infectious materials (bacteria and toxins), which are drained into the dressings, where they become dry and harmless.

Iodoform gauze has but little antiseptic action, and this action is not exerted upon the bacteria in the wound, but upon those drawn up into the gauze. The use of iodoform gauze can therefore be looked upon as an aseptic method of wound treatment.

When used as a tampon the gauze should be placed in all the recesses of the wound. In small gunshot and punctured wounds, the gauze should merely be laid over the wound and not forced into the tract, as in this way a secondary infection might be produced. Large and deep wounds with cavities, such as are produced in gunshot wounds by the explosive force of the projectile (dum-dum), should be covered with layers of iodoform gauze and the remaining spaces filled with sterile gauze to avoid the use of too much iodoform and resulting iodoform intoxication. If the deepest point of the wound is not favorably situated for capillary drainage, the tampon is often combined with tubular drainage, counter-openings frequently being required for this purpose.

In many cases the tampon must be sutured in position to prevent its displacement (in the buccal cavity, pharynx, larynx, also in the abdominal cavity).

[Cigarette drains are used very extensively in surgical work at the present time. In preparing such a drain the gauze is loosely rolled until the size required is prepared. The gauze is then wrapped in a layer of gutta percha and the drain is ready for use. The size of cigarette drains usually employed corresponds to that of the little and ring fingers. If it is desirable, tubular drainage may be combined with the capillary drainage in a cigarette drain, a piece of small rubber tubing being inclosed in the gauze. The advantage of the cigarette drain is that it can be easily removed from wounds without causing pain and injuring granulating surfaces, and it acts as efficiently as unprotected iodoform gauze packed into a wound. Iodoform or plain gauze may be used in the preparation of the cigarette drain.]

The rapid reduction of the number of bacteria in infected wounds and the prevention of progressive inflammations are usually due to the capillarity of iodoform gauze.

Moist Dressings.—The number of bacteria in a wound rapidly increases when a moist tampon or dressing is used, for example, if gauze saturated with antiseptic solutions is placed in or upon a wound (Gon-

termann). The treatment with moist compresses, evaporation from which is prevented by rubber tissue or paraffin paper, should be discarded for this reason. The bacteria multiply not only in the wound, but also in the gauze, in spite of the fact that it contains antiseptics and spread to the surrounding skin and invade the infection atria caused by maceration, producing pustules, furuncles or lymphangitis. The conditions within a moist dressing, evaporation from which is prevented, have rightly been compared to those of an incubator, and Schlange has demonstrated how bacteria will penetrate all the layers of a gauze dressing, evaporation from which has been prevented, while in dry aseptic gauze they are unable to multiply as a result of the drying of the secretion.

On the other hand, moist dressings uncovered by rubber tissue and permitted to evaporate, acquire a strong capillary action. Of course this action begins later in moist than dry dressings, which begin to absorb as soon as applied. The capillarity of unprotected moist dressings is not, however, as Nötzel thinks, greater than the dry. A greater number of bacteria may be found in the moist dressings than in the dry after a time, but the bacteria multiply in the former.

For this reason we prefer the dry dressing, or tamponade, to the moist in the treatment of infected wounds, such as acute suppurative inflammations (*vide* General Rules for the Treatment of Pyogenic Infections), and only use the moist evaporating dressings in the treatment of wounds from which is discharged a thick secretion, or where there is necrosis and the separation of the dead tissue is to be favored. In these cases the irritation of the antiseptic (preferably a three per cent solution of aluminum acetate) increases and thins the secretion, cleanses the surface, and hastens the formation of granulation tissue.

[In America warm moist dressings of a saturated solution of boric acid are used very extensively in the treatment of infected wounds, and clinical experience seems to show that they have a very favorable influence.]

Alcohol Dressings.—An alcohol compress is a good agent for cleansing wounds and infected granulating surfaces if evaporation is not prevented by rubber protective. The growth of bacillus pyocyaneus ceases if such a compress is applied three or four times, during a period of twenty-four hours. On the other hand, alcohol compresses covered with rubber protective may cause gangrene, such as frequently follows the use of carbolic acid and lysol compresses (with or without evaporation).

Carbolic Acid Compresses and Carbolic Acid Gangrene.—The moist carbolic acid compress and dressing is often used by the laity as a prophylactic measure against inflammation in accidental wounds. The harmful action of the antiseptic is best seen in cases where the dressing

has been allowed to remain some time, when not only the wound surfaces, but also the skin and deeper lying tissues may be affected. Numbness may follow after a short time the application of the compress; this numbness may later pass into complete anæsthesia. If the treatment is discontinued at this time, recovery with necrosis of the epithelium only may occur. After a longer application, and even with a one per cent solution after twenty-four hours, the whitish discoloration of the skin may pass into the black of necrosis—the tissues in contact with the carbolic acid becoming mummified. The necrosis of the skin is limited to the area in contact with the dressing. Frequently in the fingers and toes the necrosis extends deeper, involving tendons, joints, and bones, and the entire digit dies and must be amputated.

FIG. 31. — CARBOLIC ACID GANGRENE OF THE GREAT TOE, FOLLOWING THE APPLICATION OF A COMPRESS SATURATED WITH A TWO PER CENT SOLUTION OF CARBOLIC ACID FOR TWENTY-FOUR HOURS. Compress applied to a small lacerated wound.

The Use of Iodoform Gauze to Stimulate the Formation of Granulation Tissue and Adhesions. —The use of iodoform gauze is not limited to wounds from which infectious materials are to be drained away or hæmorrhage controlled. The stimulating action of iodoform causes a rapid development of granulation tissue after the tampon has been in position some days, and the formation of adhesions in serous cavities, which are of great surgical importance. By it inflammatory foci are walled off from healthy serous surfaces, and for this reason the tampon is often applied some days before deep abscesses (lung abscess or deeply situated abdominal abscess) are opened, or where the perforation of a contused part of the stomach or intestine or suture line is feared. During operations a tampon of iodoform gauze placed about the point of opening of an abscess or the point of incision of an intestinal loop protects the adjacent area from infection with pus and fæces. Iodoform gauze is usually employed in the form of doubled strips, 20 cm. in width. These can be used for practically all purposes.

The von Mikulicz Drain.—Von Mikulicz introduced into abdominal surgery the Mikulicz tampon or drain, a large quadrangular piece of gauze, the center of which is invaginated to form a pouch which may be filled with sterile gauze or drainage tubes, as the case demands.

The length of time that an iodoform gauze tampon should be allowed

to remain *in situ* depends upon the condition of the wound and the purpose for which it has been applied. In fresh wounds it may be removed after a few days, and if conditions are favorable the wound may be sutured. If the tampon has been used to control a severe hæmorrhage (e. g. in hæmophilia, or wounds of plexuses or sinuses) it should be removed only after it has been loosened by the secretion of the granulations. In serous cavities in which adhesions are to be produced, the tampon must remain at least a week.

In wounds which secrete thick pus or are beginning to granulate, and after incision of acutely inflamed tissues, the iodoform gauze should be replaced by moist compresses which are not covered by rubber tissue. The compress should be made of several layers of gauze saturated with three per cent aluminum acetate, two per cent boric acid solution or sixty per cent alcohol, and should be evenly applied.

In some cases ointments may also be used. Layers of gauze evenly arranged should be spread with a mildly stimulating or indifferent ointment (mercury, zinc, borated vaseline, or lanolin) and then applied to the wound.

Iodoform Intoxication.—Iodoform gauze may give rise to unpleasant local and dangerous general after effects. These occur rarely, and then most frequently in patients with an idiosyncrasy, or where an excessive amount of iodoform has been used. The so-called iodoform eczema rapidly spreads from the wound over an extensive area, and is accompanied by the formation of vesicles and severe itching. The edges of the wound become swollen and its surfaces coated. The itching may be controlled by zinc oxide ointment, and in most cases the healing is complete within a week. The patient's attention should be drawn to this fact, so that in later treatment the attention of the physician may be directed to this idiosyncrasy. Much more dangerous is the rare iodoform intoxication, which may develop even after careful use of the drug. It is caused by the absorption of the decomposition products of iodoform and occurs most frequently when iodoform is used in deep wounds which have not been protected from putrefactive infections (operations about rectum) and in wounds in which the reduction processes cause a rapid decomposition of the iodoform.

The symptoms in the beginning or in mild cases are persistent nausea, vomiting, and headache. These usually rapidly subside when the gauze is removed. In severe cases the symptoms, consisting of psychical disturbances, maniacal excitement, and delirium, are rapidly progressive, and are often accompanied by a cardiac weakness which may prove fatal. The urine contains iodine; often albumen and blood.

The danger of iodoform intoxication and the unpleasant odor of the powder, which is increased to an unpleasant garliclike odor by its de-

composition when coming in contact with metal (tracheotomy tube, where the dressing comes in contact with eating utensils in wounds of the hand), have led to the preparation and introduction of a number of substitutes. These may be divided into those which contain iodine, such as airol, aristol, europhen, iodol, iodoformal, iodoformin, loretin, nosophen, sozoidol, vioform, etc., and into those which do not, such as alumnol, amyloform, dermatol, thioform, xeroform, etc. The great number indicates how little satisfaction these substitutes have given, although some have found ardent supporters.

Tubular Drainage.—The drainage of a wound with glass or rubber tubes provided with lateral openings to permit of the escape of wound secretion is called tubular drainage in contradistinction to the capillary drainage obtained by the tampon. Tubes should be so placed in deep wounds as to render easy the escape of secretions from any of their parts. The outer end of the drainage tube should be provided with a sterilized safety pin or with a silk suture, which should be fastened to the skin with adhesive plaster. In this way the tube may be prevented from slipping into the wound. The pressure of the safety pin may be avoided by placing gauze between it and the skin. Often the drainage tube and tampon are combined. In this way the tube may be retained in position better and the wound kept open.

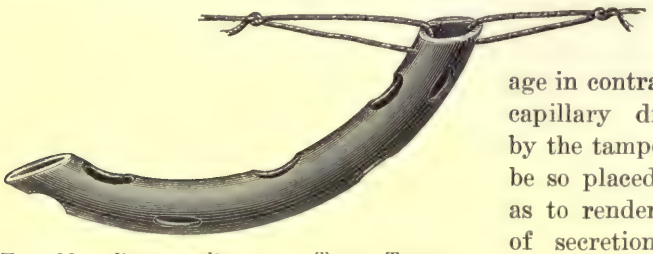


Fig. 32.—Rubber Drainage Tube, Thread Fastening.

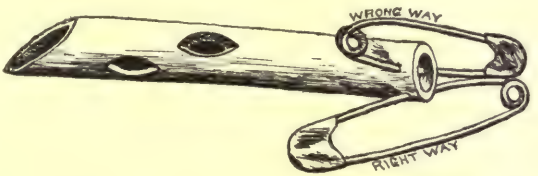


Fig. 33.—Rubber Drainage Tube, Pin Fastening.

Drainage tubes should not be allowed to remain in position longer than a few days. Their lumina easily become occluded by disintegrated blood clots and pus. When the dressings are changed new rubber drains should be inserted, glass tubes should be removed, and sterilized by boiling. As soon as the secretion diminishes and granulations become abundant, the drain may be gradually shortened, and finally removed.

Removal and Encapsulation of Foreign Bodies.—Finally the removal or encapsulation of a foreign body must be considered in the care and treatment of a wound.

All visible particles of dirt and foreign bodies should be removed from the wound with tissue or dressing forceps. If allowed to remain wound repair will be prolonged by suppuration.

A different problem confronts us when we consider the removal of deeply penetrating foreign bodies, such as needles, fragments of glass, wood and bombs, broken-off points of knives, swords, and daggers, bullets with accompanying pieces of clothing, pieces of a metal helmet, etc. According to the experiments of Messner, Brunner, and others, the bacteria upon a bullet or carried from the surface of the skin are rarely of the highly pathogenic variety. The experience of von Bergmann in the Russo-Turkish war has become highly significant in the treatment of bullet wounds, both in military and civil practice. He demonstrated that bullet wounds might pursue a perfectly normal clinical course, in spite of encapsulation of the bullet, if dry aseptic dressings were applied and the wound protected from secondary infections frequently introduced by probing, irrigation, etc. The primary infections of the tract of the wound are usually trivial and limited to its outer parts, and the bactericidal properties of the tissues overcome them.

Tetanus, putrefactive and suppurative inflammations may follow the penetration of a foreign body. In these cases the foreign body (e. g. a splinter of wood) has almost always rough surfaces, to which a great number of bacteria are attached. Inflammation may develop about an encapsulated foreign body after some years if the connective tissue capsule surrounding the foreign body and the bacteria carried in with it are ruptured by a trauma or if bacteria are deposited from the blood in the scar.

Indications and Contraindications for the Removal of Foreign Bodies.—Clinical experience has established the following fundamental principles in the treatment of penetrating foreign bodies.

Foreign bodies should be removed:

1. If they are visible in the wound.

2. If the foreign body, such as a splinter of wood, has rough surfaces, and is frequently followed by inflammation or tetanus.

3. If the foreign body can be felt directly beneath the skin and its removal is a trivial procedure.

4. If the foreign body immediately, or later after being displaced by muscular action, presses upon or is situated within a nerve, if it irritates mucous or synovial membranes, or causes pain by its point or sharp surface when pressed upon during movement (as needle, fragment of glass, etc.).

5. If a phlegmon or tetanus develops in the tissue surrounding the tract of the foreign body.

Unless there are positive indications no effort should be made to

locate the foreign body with a probe or remove it with forceps, as there is danger of introducing secondary infection. Incisions for the removal of foreign bodies should be made some distance from the wound if it is infected or should be delayed until healing has occurred.

The position of metallic foreign bodies and glass may be accurately determined by the use of the X-ray, at least two views from different known angles being necessary. Information can also be gained by palpation, location of the pain, and the disturbance of function.

Encapsulation is to be encouraged in all cases in which the foreign body is smooth and deeply situated, gives rise to no disturbance, and in which there is no inflammation of the tract of the wound.

If after encapsulation there is pain or interference with function, the benefits to be derived from an operation are to be carefully weighed against the gravity and dangers of the same (for example, foreign body in the brain or vertebral column or in the thorax).

The most important rules for the treatment of accidental wounds may be shortly summarized as follows:

Emergency Dressing.—Immediate covering of the wound with dry sterile gauze.

Definitive Dressing.—Cover the wound with sterile gauze while the surrounding area is being sterilized. If hæmorrhage is severe, apply Esmarch's elastic constrictor. Preparation of area about wounds, the same as the field of operation in an aseptic procedure. Cover the surrounding area with sterile towels. Anæsthesia if necessary and not contraindicated. Cautious separation of the edges of the wound with retractors to permit of inspection. Removal of dirt and foreign bodies with forceps, of blood clots with gauze. Irrigation of dirty wounds with a three per cent solution of hydrogen peroxide, application of artery forceps and ligation of vessels, removal of fragments of tissue, trimming off of contused edges of wounds, tampon, drainage or suture, dry aseptic, and immobilizing dressing.

LITERATURE.—*Bass.* Erfolge und Gefahren der Gelatineapplikation. Zentralbl. f. d. Grenzgebiete, 1904, p. 818.—*Bierfreund.* Ueber den Hämoglobingehalt bei chir. Erkrankungen, mit besonderer Rücksicht auf den Wiederersatz von Blutverlusten. Chir.-Kongr. Verhandl., 1890, II, p. 159.—*Brunner.* Ueber die Infektion der Schusswunden durch mitgerissene Kleiderfetzen. Korresp.-Blatt f. Schweiz. Aerzte, Bd. 26, 1896.—*v. Esmarch.* Ueber künstliche Blutleere. Chir.-Kongr. Verhandl., 1896, II, p. 1.—*Gontermann.* Experim. Untersuchungen über die Ab- oder Zunahme der Keime in einer accidentellen Wunde unter rein aseptischer trockener u. antiseptischer feuchter Behandlung. Arch. f. klin. Chir., Bd. 70, 1903.—*Heile.* Ueber die antiseptische Wirkung des Jodoforms. Chir.-Kongr. Verhandl., 1903, II, p. 376.—*Honsell.* Experim. u. klin. Untersuchungen über die Verwendbarkeit des Wasserstoffsuperoxydes. Beitr. z. klin. Chir., Bd. 27, 1900, p. 127.—*A. Köhler.* Transfusion u. Infusion seit 1830. Gedenkschr. f. v. Leuthold. Berlin, 1906, Bd. 2, p. 271.—*Kronecker u. Sander.* Bemerk. über lebensrettende Transfusion von anorgan, Kochsalzlösung. Berlin. klin. Wochen-

schr., 1879, No. 52.—*Küttner.* Zur Frage des künstlichen Blutersatzes. Chir.-Kongr. Verhandl., 1903, I, p. 24;—1st die physiol. Kochsalzlösung durch die Tavelsche Sodasalzlösung zu ersetzen? Beitr. z. klin. Chir., Bd. 35, 1902, p. 272.—*Landois.* Blutverlust, Transfusion. Lehrb. d. Physiol. d. Menschen.—*Leonpacher.* Ueber Kochsalzinfusion. Mitt. aus d. Grenzgebieten, Bd. 6.—*Lossen.* Die Bluterfamilie Mampel. Deutsche Zeitschr. f. Chir., Bd. 75, 1905, p. 1.—*Medizinalabteilung* des k. preuss. Kriegsministeriums, Ueber die Wirkung u. kriegschir. Bedeutung der neuen Handfeuerwaffen, Berlin, 1894.—*Messner.* Wird das Geschoss durch die im Gewehrlauf stattfindende Erhitzung sterilisiert? Münch. med. Woch., 1892, p. 401.—*Nötzel.* Experim. Studie zum antisept. Wundverband. Arch. f. klin. Chir., Bd. 71, 1903, p. 165. —*Perman.* Die Angiotripsie in der operativen Chirurgie. Zentralbl. f. Chir., 1904, p. 1098.—*Pfuhl.* Ueber die Infektion der Schusswunden durch mitgerissene Kleiderfetzen. Zeitschr. f. Hygiene, Bd. 13, 1893, p. 487.—*Schimmelbusch.* Anleitung zur asept. Wundbehandlung, Berlin, 1893.—*Schlange.* Ueber sterile Verbandstoffe. Chir.-Kongr. Verhandl., 1887, II, p. 141.—*Stempel.* Die Hämophilie. Zentr. f. Grenzgebiete, 1900, No. 18.—*Strubell.* Der Aderlass, Sammelreferat mit Lit. Zentr. f. Grenzgebiete, 1903, p. 1.—*Zimmermann.* 6 Fälle von Hautgangrän nach subkutaner Infusion von Kochsalzlösung. I.-D. Tübingen, 1900.

CHAPTER III

WOUND REPAIR

THERE are two methods of wound healing. If the surfaces and edges of wounds are closely approximated or held in contact by sutures union occurs within a few days. If, on the other hand, the wound gapes or there is an actual loss of substance, new tissue must be formed to fill in the defect. The covering of this new tissue with epithelium completes the process of healing. Healing by the first method is called primary wound healing or healing *per primam intentionem*; by the second method, secondary wound healing or healing *per secundam intentionem.*

Primary Wound Healing.—*Primary healing* occurs, if not prevented by suppurative inflammation, necrosis, or the exudation of blood, when the edges of the wound are approximated. The union of a superficial incised wound or of a sutured wound requires from a week to ten days. When the crust which covers the line of union and the desquamated epithelium of the edges of the wound fall off, a delicate reddish epithelial membrane covering the space between the edges of the wound is seen. Gradually the newly formed epithelium cornifies and the firm, red scar becomes soft and white. The white color and smooth surface of the scar are permanent. Only very superficial scars disappear completely after some years.

Primary wound healing is more complete and rapid if the edges of the wound are accurately approximated, but even then it is a complicated process. Only in wounds of the epithelium do we find a direct union of the edges by newly formed cells which replace those injured and destroyed. In all vascular tissues such a union by simple regeneration is impossible. Blood and tissue fluids which are poured out into the wound prevent, even in smallest amounts, the approximation of the wound edges, and besides, in all, even incised wounds, the tissues are injured considerably by the trauma, and whole groups of cells are destroyed by the subsequent circulatory and nutritional disturbances, by the exposure to the air, and, if the wound has been improperly treated, by the contact with water and antiseptics.

The accumulation of blood and tissue fluids and the death of tissues prevent a direct union, but incite processes which provide for heal-

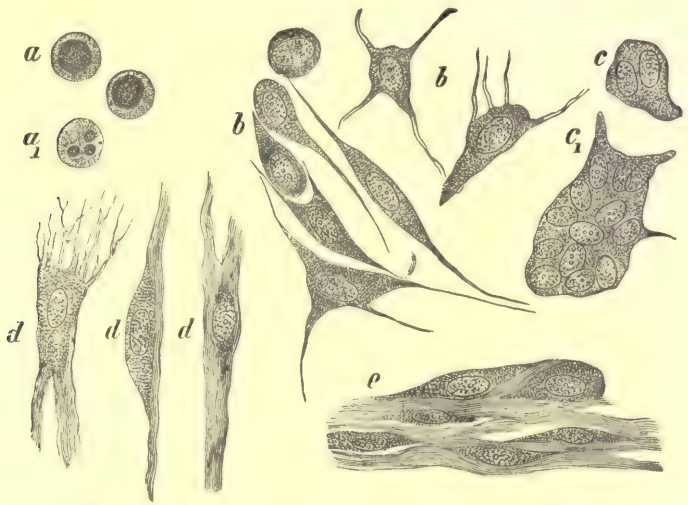


FIG. 34.—ISOLATED CELLS FROM GRANULATION TISSUE. *a*, Lymphocytes or mononuclear leucocytes; *b*, different forms of mononuclear connective tissue cells; *c*, polynuclear connective tissue cells; *d*, connective tissue cells forming fibrillæ; *e*, fully developed connective tissue. (After Ziegler.)

ing of the wound. The irritation resulting from the trauma, secondary injuries, and degenerating cell masses gives rise to the symptoms of inflammation which, because of its etiology, is known as *mechanical* or *traumatic* inflammation.

During this stage the edges of the wound become united by a layer of fibrin which is formed by the coagulation of the blood, lymph and wound secretion. (Agglutination is the first step in primary wound healing; the earlier it begins, the more rapid and complete the union.)

Even when a wound heals by primary union, granulation tissue, the germinal tissue which fills in the defect, is formed, but in minimal amounts.

During the process of agglutination the neighboring blood vessels become congested. There is a peripheral stasis and emigration of leuco-

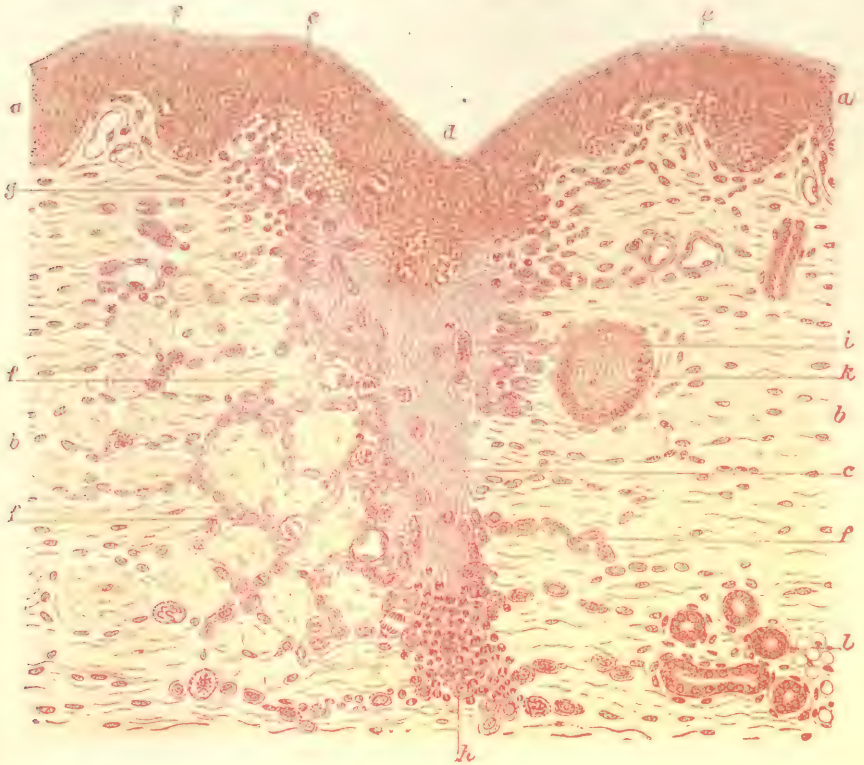


FIG. 35.—HEALING OF A SUTURED INCISED WOUND OF THE SKIN SIX DAYS OLD. (After Ziegler.) *a*, Epidermis; *b*, corium; *c*, fibrinous part of the exudate; *d*, newly formed epidermis which contains numerous karyokinetic figures and epithelial processes which have penetrated the exudate beneath it; *e*, karyokinetic figures in the epithelium somewhat removed from the line of incision; *f*, germinal tissue developing from the connective tissue which also contains proliferating vessels; *g*, proliferating germinal tissue with leucocytes; *h*, groups of leucocytes in the inner part of the wound; *i*, fibroblasts lying in the exudate; *k*, sebaceous glands; *l*, sweat glands.

cytes, and some exudation, which assists in the formation of the fibrin layer. The accumulation of polymorphonuclear leucocytes, which can be demonstrated in from three to four hours, is well marked in twenty-four hours (Marchand). The number of leucocytes in the wound depends upon the amount of injury and degeneration.

Functions of Leucocytes.—These cells perform many functions. They secrete a ferment which digests albumen and liquefies the degener-

ating tissues and they produce bactericidal bodies, which destroy the pathogenic bacteria. The latter are attenuated in aseptic operation- and accidental wounds (Schloffer, Riggenbach, Brunner). Leucocytes have phagocytic properties also. Their protoplasm is motile, and they are able to surround and ingest particles of tissue and the products of degeneration. The young fixed tissue cells (tissue-phagocytes or macrophages) rich in protoplasm are more actively phagocytic than the leucocytes. These ingest fat and pigment granules and become transformed into the so-called fat or pigment granule globules, which may pass through the lymph stream into the glands. The leucocytes rapidly degenerate and are replaced by new cells; often they are found in the cytoplasm of the large phagocytes.

Proliferation of Fixed Tissue Cells.—The proliferation of the fixed tissue cells goes hand in hand with the changes above described. Numerous karyokinetic figures indicate the activity of growth in the deep layers of the epithelium, in the endothelium of the vessels, and in the fixed tissue cells. Large cells of different forms with one, two, or many nuclei (the latter are called giant cells) grow from the tissues into the wound cleft. These cells are the formative connective tissue cells and are called fibroblasts. They infiltrate the agglutinating layer of fibrin, and later form the fibrillæ of the new connective tissue, which holds the edges of the wound together and is known as a scar.

Formation of New Blood Vessels.—This connective tissue is composed not only of fibroblasts, but also of leucocytes, lymphocytes, and plasma cells (*vide* Inflammation, Part II). The formation of new blood vessels accompanies the proliferation of fixed tissue cells, and is the result of the actual sprouting of solid, arched, protoplasmic processes from the walls of preëxisting vessels. These protoplasmic processes later become united with each other, and their interior becomes liquefied, hollow, and patent.

Time Required for Healing of Clean Incised Wound.—A clean-cut wound with an undisturbed clinical course heals, as a rule, in about one week or ten days. With the development of the fibrillar ground substance, the newly formed scar, which in the beginning is vascular and rich in cells, contracts. It becomes paler and narrower. The firmness of the scar is gradually lost as the newly formed connective tissue bundles separate. The scar can be distinguished microscopically from the surrounding tissue for some time by its firmer texture and by the absence of elastic fibers. The less the amount of injury, and therefore the less the inflammation, the finer and more nearly perfect the scar will be. Broader and more resistant scars remain after the primary healing of contused and lacerated wounds than after the primary healing of incised wounds.

Secondary Wound Healing.—Secondary wound healing, characterized by the development of a reddish, granular tissue which bleeds easily, differs from primary wound healing. It depends upon the same, but

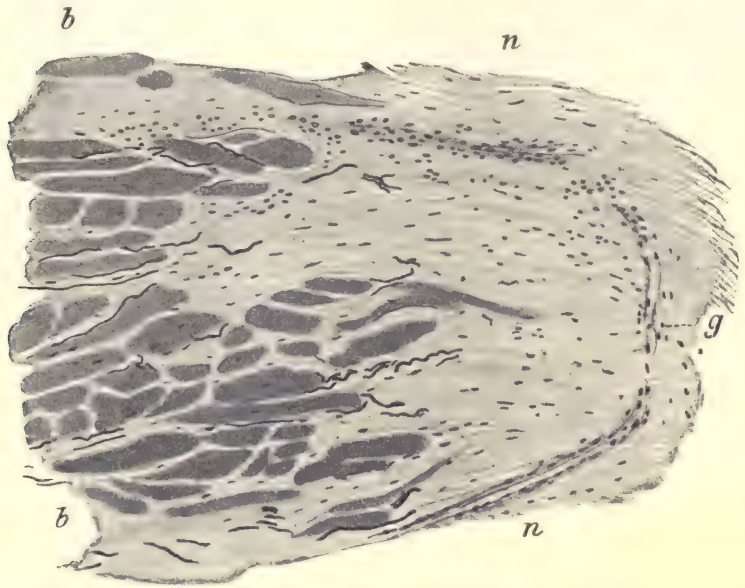


FIG. 36.—SECTION FROM A SCAR IN THE SKIN TWENTY-SIX DAYS OLD. (After Marchand.) The scar (*n*) consists of a fibrillar tissue containing numerous oval nuclei of connective tissue cells and newly formed blood vessels; (*g*) the connective tissue bundles of the cutis are intimately interwoven with the newly formed tissue. Some of the old elastic fibers pass beyond the margins of the scar.

considerably exaggerated, processes. The germinal tissue—called granulation tissue, because of its granular appearance—lies exposed in the wound, fills all the spaces of the wound, and replaces the lost tissues. This granulation tissue develops in all wounds in which primary wound healing has been prevented by the accumulation of blood or wound secretion or by extensive necrosis of the tissues following trauma or infection.

Soon after the injury a layer of fibrin mixed with some blood forms upon the surface of the wound. Within two days this fibrin layer becomes quite firmly attached to the hyperæmic swollen tissues of the wound and transformed into a yellowish, cheesy, fibrino-purulent membrane as a result of superficial necrosis or bacterial inflammation accompanied by the accumulation of leucocytes. The secretions discharged from such a surface may present all the transitional forms between the serous and sero-purulent. Earliest after three days, often after one week, the membrane becomes penetrated at different points by small red

granules, each granule corresponding to a small blood vessel surrounded by germinal tissue. Finally the necrotic particles are separated and cast off by this tissue, and the entire surface of the wound covered by it.

Healing of granulating surfaces may be hastened by drawing the edges of the defect together with adhesive strips or inserting tension sutures. Where the granulating surfaces come in contact they will unite.

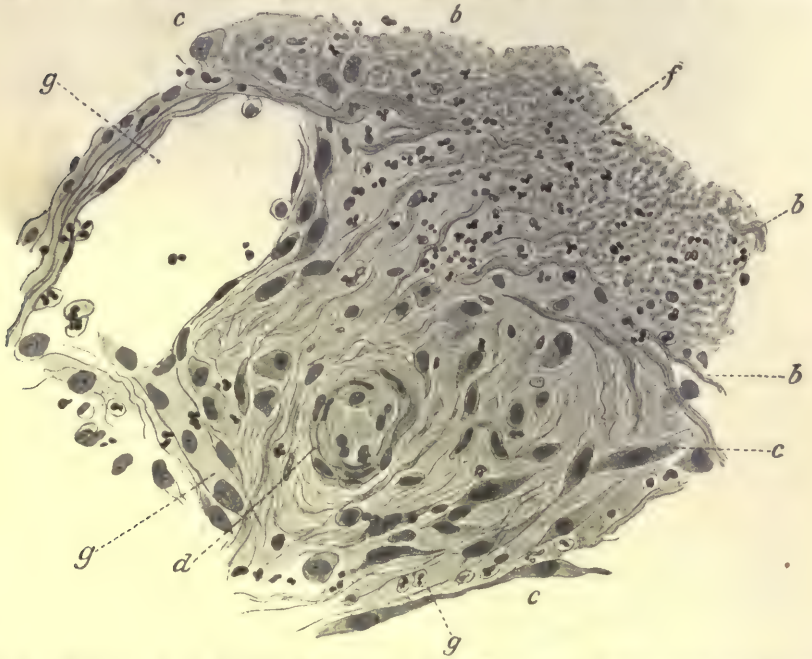


Fig. 37.—Section from Wound Four Days Old, Following Amputation of a Dog's Tongue. (After Marchand.) The ends of the divided connective tissue bundles (*b*) extend into the layer composed of fibrinous exudate *f*; within and beneath the fibrinous exudate are numerous polynuclear leucocytes; *g*, small vessel which becomes continuous with a dilated blood space; *d*, small artery with proliferating wall; *c*, enlarged fusiform, irregular connective tissue cells.

Granulation tissue secretes an exudate rich in cells which resembles pus. This exudate, if it does not dry and form a crust, cleanses mechanically the surface of the wound, and has a bactericidal action (cf. Pyogenic Infections, Part II, p. 155). It is especially profuse if the tissue is infected.

The fibrillæ formed by the fibroblasts are arranged parallel to the surface in the deeper parts of the wound. From here they pass vertically along with the vessels (Figs. 37 and 38) into the upper, but less dense, layers.

The covering over of a granulating surface with skin proceeds gradually from the margin of the wound in the form of a bluish border. The

new epithelium grows into the depths between the vascular loops of the granulations. Small islands of epithelium also develop in the center of the granulating surfaces from the ducts of sweat glands and from hair follicles which were not totally destroyed by the injury or infection.

Thick connective tissue bundles form as the scar develops. By the contraction of these bundles the scar is reduced in size and neighboring structures are often drawn out of place, causing unsightly deformities (ectropion of the lids and lips). Movements of the fingers, toes, and larger parts of the extremities may be interfered with (cicatricial contractures). Elastic fibers, nerves, and the appendages of the skin are not found in the scar.

The contracting scar becomes extremely pale as the vessels become obliterated. The surface of a scar is smooth and white and remains so. After the loss of a large amount of tissue the scar may be depressed.

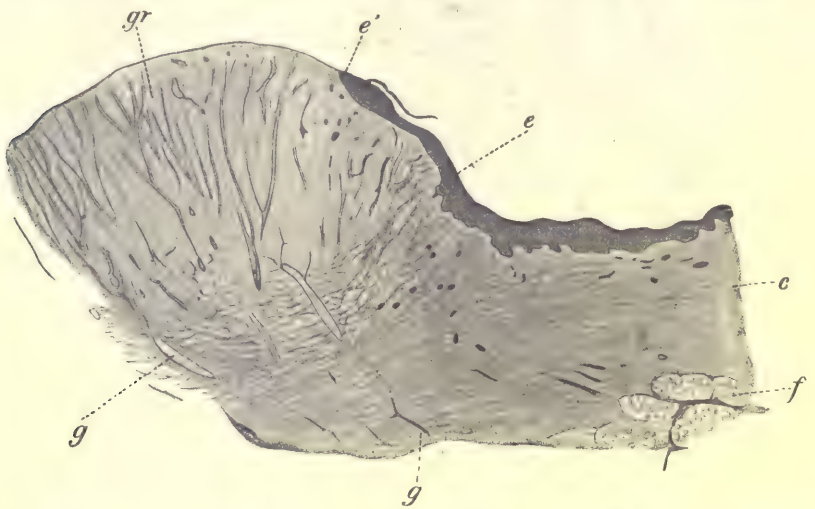


FIG. 38.—SECTION THROUGH THE EDGE OF A GRANULATING INCISED WOUND ABOUT THREE WEEKS OLD SURROUNDING A FISTULÆ LEADING TO AN OSTEOMYELITIC FOCUS IN THE FEMUR. (After Marchand.) *c*, Cutis; *f*, fat; *e*, *e'*, newly formed epidermis; *gr*, granulations; *g*, vessels of the granulations ascending from the cutis.

Hypertrophic Scar, Cicatricial Keloid.—If the connective tissue continues to develop and does not contract, large tumor-like masses of cicatricial tissue supplied with large blood vessels form (hypertrophic scar, cicatricial keloid).

Healing Beneath a Scab.—Healing beneath a scab sometimes resembles more closely primary, at other times, secondary wound healing. It is seen most often in non-penetrating, superficial wounds of the skin (excoriations of the skin, defects following removal of strips of skin for grafting). The blood and tissue fluids dry to form a firmly attached

crust, or become united with the dressings to form a protective covering. Beneath this covering, if it is not forcibly removed, and if inflammation

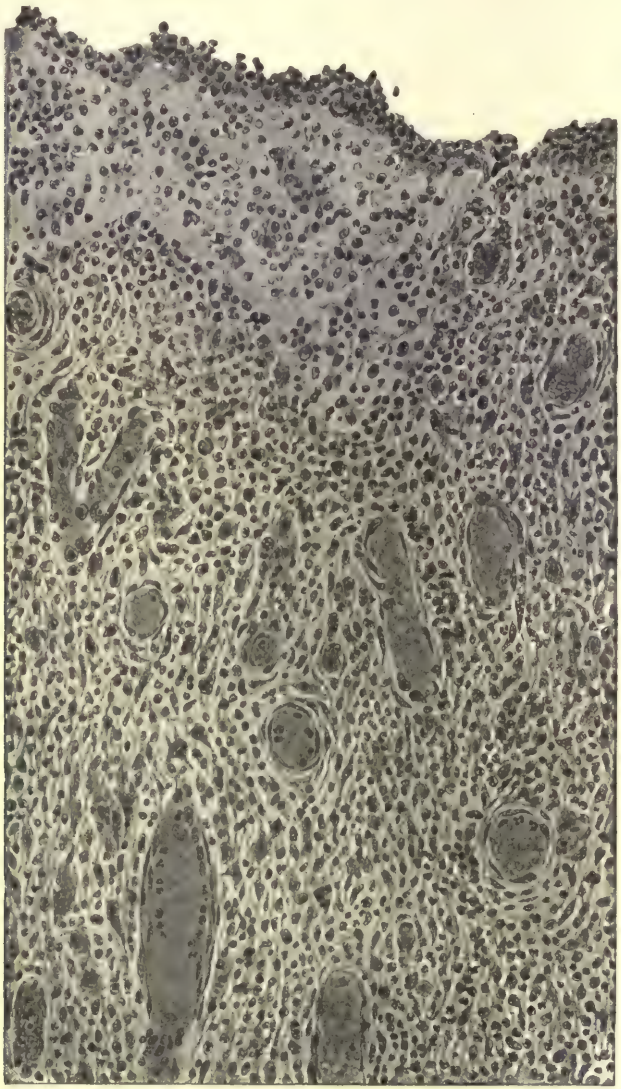


FIG. 39.—SECTION OF GRANULATION TISSUE REMOVED FROM AN ABSCESS MEMBRANE SURROUNDING A SUPPURATING FOCUS IN BONE. Superficial layer.

does not occur, new epithelium which develops from the edges of the wound and from the deeper layers of the stratum Malpighii, in the same way as in burns of the second degree, is formed in a few days.

In deep wounds the growth of epithelium, which then develops only from the edges of the wound, may go on to completion beneath the scab, which in a fresh wound is formed by the wound secretion and the firmly adherent gauze, in deep contusion of the skin by necrotic tissue, and after cauterization by the layer of tissue which has been destroyed. The epithelium extends beneath the scab and covers the wound, if the clinical course is undisturbed, before granulation tissue develops in large amounts. After the surface is covered with epithelium the crust falls off and the dry gauze which may have been applied becomes separated.

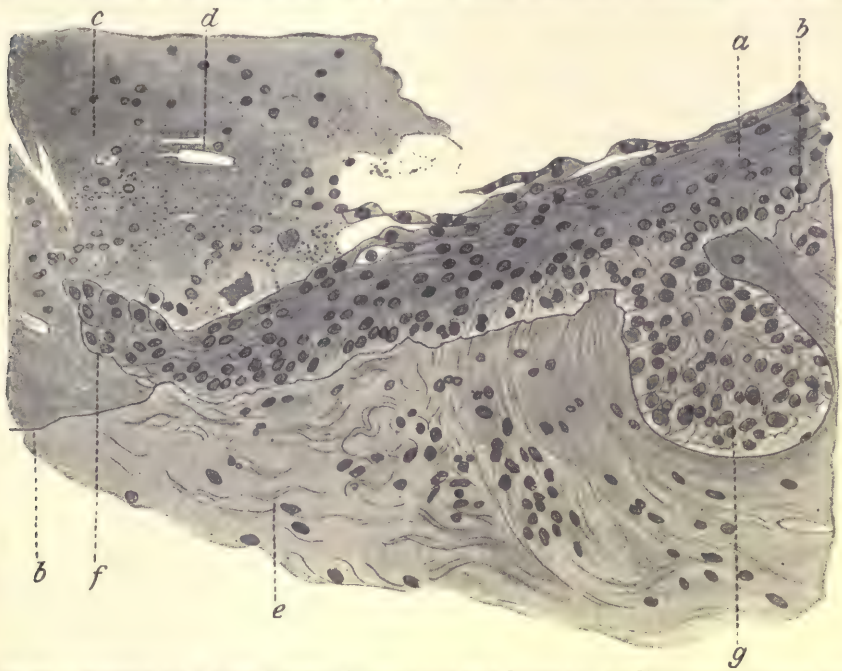


FIG. 40.—MARGIN OF A SKIN DEFECT FOLLOWING THE REMOVAL OF EPIDERMAL STRIPS FOR SKIN GRAFTING. (After Marchand.) *a*, Newly formed epidermis at the margins of the wound; *f*, end of the same which projects in the form of a process into the crust; *c*, which is somewhat loosened; *d*, space in which iodoform crystals lie; *e*, cutis; *f*, which is almost unchanged; *g*, a newly formed epidermal growth which has extended downward in the form of a round process between the connective tissue bundles.

If healing beneath the scab is disturbed by removal of the crust, or dressings, or by suppuration, granulation tissue develops in relatively large amounts if the wound has not already become covered by epithelium.

Occasionally granulating surfaces may be covered by epithelium, which develops under the scab formed by the desiccation of the secretion discharged from them. Usually, however, such scabs retard healing, as they prevent the discharge of wound secretion.

Macroscopic Appearance of Granulation Tissue.—Exposed granulation tissue varies in appearance and behavior, depending upon whether it is healthy or diseased, normal or pathological. Healthy granulation tissue is granular, firm, deep red in color, bleeds easily, and secretes little. Diseased granulation tissue is pale, soft, spongy, its surface smooth, and the granulations are covered by a membrane.

Causes of Unhealthy Granulation Tissue.—The cause of these pathological changes in granulation tissue may be local or general. In anæmia, cachexia, syphilis, and diabetes the organism is rarely able to form good, healthy granulations. They are pale and flabby, and as little inclined to form connective tissue as the epithelium is to cover over the granulating surface. The local causes are usually infectious. If the surface of the granulating tissues contains microbes, which multiply in its fibrinous secretion, or is continually bathed in pus, as in fistulous tracts or in areas adjacent to dead tissues, the newly formed granulations may grow luxuriantly, even about the level of the edges of the skin (proud flesh, caro luxurians). If this is the case connective tissue is not formed, as the fibroblasts are destroyed by the bacterial toxins (Reinbach). If the membrane containing the bacteria is removed by an increased exudation following the artificial irritation (viz., quicksilver salve, silver nitrate), or the deep necrotic tissue (tendon, piece of bone) is removed, the flabby, pale granulations will become transformed into healthy granulations and healing will occur. Covering over of unhealthy granulations with skin is impossible, partly because of the luxuriant growth of the granulations above the edges of the skin, and partly because of the secretion of pus from the infected surface.

Granulating Wound and Ulcer.—The granulating wound resulting from an injury, a burn, or cauterization is distinguished from the granulating ulcer, in which there is a progressive destruction of tissue. The ulcer heals only after the unhealthy granulations have been transformed into the healthy (viz., tuberculous, syphilitic, tropho-neurotic ulcers).

The Repair of Different Tissues.—In tissues composed of connective tissues and specific elements, repair by granulation tissue or scar tissue is the most important method. Regeneration of the specific cells plays a secondary and subordinate rôle (muscle, nerve, tendons, large viscera). In some cases these specific elements do not regenerate at all (brain, also apparently spinal cord, *vide* Part IV). Cartilage and bone repair by the formation of germinal tissue, which develops from the perichondrium and periosteum, the germinal tissue reproducing the same type of tissue as that destroyed (callus). The union of two serous surfaces, which are approximated in intestinal suturing, begins with agglutination by a layer of fibrin, and is completed by the formation of granula-

tion tissue, which also unites the remaining layers of the intestinal wall. The scar upon the inner surface of the intestinal wall is covered by new epithelium in which gland tubules form; there is but little regeneration of the smooth musculature. Wherever the serosa is lost or destroyed a layer of fibrin forms which later becomes infiltrated with granulation tissue, leading to the formation of adhesions, therefore the peritoneum in all abdominal operations must be spared as much as possible and protected from all mechanical, chemical, and physical (e. g., desiccation) insults. Similar adhesions may be caused by blood clots, which become attached to the peritoneum or by inflammatory exudates.

Injured and ligated arteries are repaired by the formation of scar tissue. The proliferation of the endothelium and connective tissue of the vessel wall, with the formation of new blood vessels which develop from the vasa vasorum or adjacent blood vessels, precedes scar formation. If a thrombus develops at the point of injury, as in contusions or lacerations or where the intima is roughened (arterio-sclerosis) repair will be delayed by organization of the thrombus. When vessels are ligated under aseptic precaution, a thrombus does not form as a rule; if it does it is small. The connective tissue which in some weeks absorbs the catgut and replaces or encapsulates silk ligatures, contributes to the firmness of the scar, which usually in large vessels is sufficiently resistant in about three weeks to withstand the blood pressure unassisted by ligatures.

Digestion, Extrusion, Encapsulation of Foreign Bodies by Granulation Tissue.—Granulation tissue also plays an important rôle in the digestion, extrusion, and encapsulation of foreign bodies and necrotic tissue and in the healing of completely separated but viable tissues (transplantation).

These essentially different processes depend upon similar tissue changes. The irritation of the foreign body causes a reaction which stimulates the growth of germinal tissue. Leucocytes and fibroblasts ingest absorbable and dead material, and produce lacunæ about the periphery of a foreign body which are comparable to the lacunæ produced by osteoclasts in the absorption of bone. Fibrin and catgut are completely absorbed by cellular activity and replaced by proliferating connective tissue. Necrotic is loosened from healthy tissue by the same process and surrounded by a wall of connective tissue, and is finally completely separated. If the secretion of the surrounding granulation tissue perforates the skin, and the opening is favorably situated, the separated dead tissue (necrotic tendon, sequestrum of bone) will be extruded. It is the same with penetrating foreign bodies which have carried bacteria into the wound. The rapidly growing connective tissue surrounding such a body cannot cicatrize, but maintains a profuse puru-

lent secretion which prevents healing of the wound and hastens the extrusion of the foreign body. On the other hand, foreign bodies which irritate the tissue but little, and contain only bacteria which are readily destroyed by the tissue, are completely encapsulated and the wound heals. Such foreign bodies become imbedded in a connective tissue capsule. The encapsulation of bullets, pieces of steel, and especially of suture material (silk, silver, aluminum bronze wire) is a very important process from a surgical viewpoint. Buried silk sutures become completely infiltrated with germinal tissue and can be used to bridge over defects in the repair of tendons, the germinal tissue developing between the sutures and bridging over the defect.

Virulent bacteria may be encapsulated with foreign bodies, from which deep inflammatory processes may develop years later if the surrounding connective tissue capsule is ruptured by trauma. Frequently pieces of clothing which have been carried into the tissues with bullets become encapsulated.

Skin Grafting.—Skin grafting is employed to promote the rapid healing of large fresh wounds and granulating surfaces of different sizes. A number of different methods have been devised, but only those will be mentioned which have been tested and found to be satisfactory. In the method devised by Reverdin and Thiersch small pieces or long strips of epidermis including the stratum papillare are used, while in the method employed by von Esmarch, Krause, and others the entire cutis (cutis strips) with or without a layer of fat is employed. The raw surfaces of the grafts are applied to the granulating or vivified wound surfaces.

Method of Repair.—As in primary wound healing, agglutination by a thin layer of fibrin is the first step in the process of healing of skin grafts unless prevented by hæmorrhage or infection. This fibrin layer is soon infiltrated by leucocytes, fibroblasts, and newly formed vessels developing from the wound surfaces. Circulation is reëstablished in the grafts in a short time. Enderlen has successfully injected these newly formed vessels and traced them into the papillary layer. He has demonstrated their presence in epidermal grafts as early as the second day; in cutis grafts as early as the third day. In the epidermal grafts only the superficial epithelium dies; the deeper layers proliferating actively as early as the second day. The entire epidermis is cast off from cutis grafts, but is replaced by the proliferation of islands of epithelium remaining in the deeper layers of the epithelium, of the sweat glands and of the edges of the wound.

Epidermal grafts heal in position more rapidly than cutis grafts. When the former are used, repair is definitely established in from one to two weeks, while the latter require from three to five weeks. W.

Braun states that the fibrous and elastic elements of the grafts are retained. His findings are the opposite of those of Enderlen, and he believes that this is due to the fact that he had more favorable material for examination.

Histological Changes in the Grafts.—According to the histological investigations of Enderlen, the fibrous and elastic tissues of the cutis grafts gradually degenerate and are replaced by newly formed tissue, which develops in part from the connective tissue elements of the graft. The cells of the ducts of sweat glands and hair follicles also regenerate to replace those lost during the first few days.

Sensation is restored in the graft in about six weeks, extending from the periphery toward its center.

The changes occurring in the pigment of the skin are interesting and remarkable. If skin is transplanted from a negro to a white man, the pigment gradually disappears and the graft becomes white, while if the graft is taken from a white man and placed upon a negro, it gradually becomes pigmented.

Early Appearance of the Grafted Area.—The grafted area appears bluish red in color and slightly depressed at first. Gradually the color becomes paler, and as contraction occurs a smooth scar, covering the site of the former defect, develops; sometimes, however, disfiguring keloid-like masses develop upon the surface. The wound resulting from the removal of the grafts heals in from one to two weeks under a dry dressing (healing beneath a scab), and after this time grafts may again be taken from the same area, which remains of a reddish color for some time and later becomes pigmented.

Transplanted cutis strips contract but little, when the process of healing is undisturbed; contracting the least when they contain a thin layer of fat. In about five weeks they resemble closely the surrounding skin, are movable, and are easily displaced over the subjacent tissue (*vide* Plastic Operations). The healing of secondary defects may be hastened by suture and the grafting of epidermal strips.

Transplantation of Mucous Membrane.—The transplantation of mucous membrane was successfully attempted by Czerny in 1871. It has been used particularly by Wölfler, Uhthoff, and others to repair conjunctival defects and replace eyelids; the mucous membrane being taken preferably from the lips or cheeks. If the epithelium of the grafts desquamates, it is rapidly regenerated.

Transplantation of Cartilage and Bone.—Cartilage may be successfully transplanted if the perichondrium is attached. Kredel used a piece of the auricular cartilage to support the alæ nasi; von Mangoldt introduced subcutaneously a costal cartilage to raise a sunken nasal bridge; Fritz Koenig used with excellent results a cuneiform piece of

the pinna to replace the alæ nasi. If the perichondrium is not transplanted the cartilage is gradually absorbed (Marchand).

The transplantation of bone to overcome a bony defect is of great surgical importance. Attempts had been made at the beginning of the last century to close trephine openings, by replacing the button of bone removed. The experimental and practical work of Ollier has extended the usefulness of bone transplantation in a number of different ways.

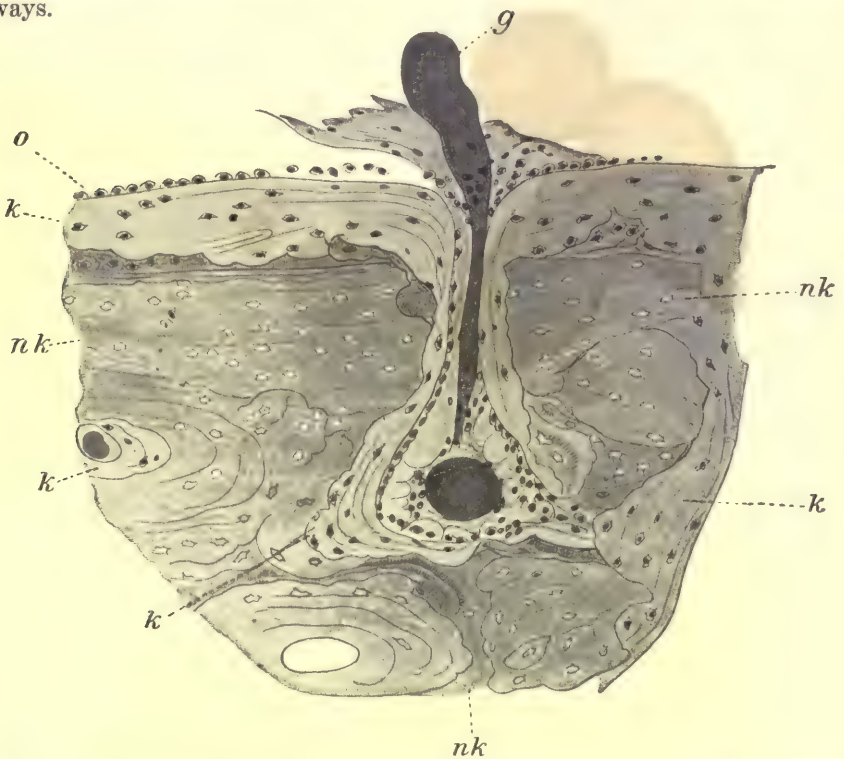


FIG. 41.—BONE FORMATION AT THE MARGIN OF A MEDULLARY CAVITY AND ABOUT THE HAVERSIAN CANALS (FRESHLY TRANSPLANTED BONE AS IT APPEARS MICROSCOPICALLY AFTER FORTY-NINE DAYS). (After Marchand.) *o*, Osteoblasts; *k*, newly formed bone; *nk*, bone which has become necrotic; *g*, an injected blood vessel.

It makes little difference whether the bone is transplanted with or without periosteum and medulla, whether it is taken from the patient, from another person, or from a lower animal, whether it is living or dead (and in the latter case sterilized by boiling or flaming). Bone differs in this respect from all other tissues. Barth and Marchand offer the following explanation of this difference: Transplanted bone is never completely preserved, being for the most part absorbed and replaced by newly formed bone. The salts of bone play an important rôle in its

regeneration, for while decalcified bone is quickly absorbed, the bone ash stimulates the encapsulating connective tissue to form thin lamellæ of bone (Barth). Pieces of bone with periosteum attached, which are taken from the same person and immediately transplanted, offer conditions most favorable for encapsulation.

In the process of encapsulation a layer of fibrin is formed first which later becomes infiltrated by newly formed connective tissue. The nuclei of the bone corpuscles and marrow cells degenerate and they die, only the most superficial layers of cells in the transplanted bone being preserved, unless injured during transplantation. As early as the fifth day young connective tissue cells, developing from the adjacent actively proliferating connective tissue, and young blood vessels grow into the medullary spaces and Haversian canals. On the eighth day, as a rule, the entire piece of transplanted bone is surrounded and infiltrated by connective tissue, the replacement of the dead medullary tissue in the interior of the transplanted bone requiring a longer time.

[The transplanted bone is gradually absorbed, being replaced by granulation tissue which eventually forms new bone. The transplanted bone then acts merely as a scaffolding or framework for the rapidly proliferating bone tissue. These newly formed cells infiltrate the Haversian canals and the bone marrow and aid in the absorption of the transplanted tissue. Giant cells are also found, especially upon the surface of the transplanted bone, which correspond to the osteoclasts found in normal bone formation. These giant cells also perform apparently the same function in transplanted as in developing bone, digesting the bone and aiding in its removal. The giant cells usually lie in deep depressions upon the surface, which correspond to Howship's lacunæ. The more rapidly this granulation tissue forms, the more rapidly the layers of bone containing degenerated nuclei are destroyed and replaced. The space between the transplanted bone and the edges of the bone is soon filled with this tissue.]

The time required for absorption and replacement usually depends upon the thickness of the piece of bone transplanted, being most rapid when fresh material is used. If some of the periosteum about the bony defect is raised and laid over the transplanted bone, repair will be hastened.

The Use of Ivory Pegs to Fix Fragments of Bone.—Ivory, which is used mostly in the form of pegs to unite fractures, is acted upon in much the same way as dead bone tissue. The surface of the ivory peg is gradually worn away and the small depressions are filled with newly formed granulations, which are firmly united with the surrounding tissue, and the peg is eventually encapsulated if sterile.

Transplantation of Muscles and Nerves.—Strictly speaking, muscles and nerves are not used for transplantation. If separated from their

connections they degenerate completely, even if healing occurs. By muscle, tendon, and nerve transplantations are understood operations in which functioning, living structures are united with diseased, nonfunctioning structures; the connections of the living tissue, however, never being completely divided (cf. Injuries and Diseases of Soft Tissues).

Czerny used successfully a lipoma to replace a breast which he had amputated for an adeno-fibroma and hypertrophy.

The transplantation of parts of blood vessels has been successfully performed by Hoepfner and Carrel and Guthrie in animals. Hoepfner used the technic advised by Payr in making the arterial anastomoses (cf. Injuries of Arteries). The defect in the artery was replaced by a piece of an artery from the same animal or another of the same species.

The transplantation of parts of organs is of practical importance. Thyroid gland tissue has been transplanted in cases of myxœdema following operations, and although encapsulation with regeneration is possible, the results are not satisfactory. Feeding of thyroid preparations is more simple and just as effective.

The Use of Foreign Inorganic Materials.—Foreign inorganic materials (alloplasty) have been used for some time to close defects in bone, particularly in the skull, to raise the sunken nasal bridge in saddle-nose, or to close large hernial rings. Plates of amber, platinum, celluloid, ivory, gold foil, and silver wire filigree are inferior to living bone for purposes of transplantation. These foreign bodies will become encapsulated if they have been previously thoroughly sterilized, and if the operation is performed aseptically. If, however, they produce pressure upon or rub against the skin, fistulæ will form. Liquid or solid paraffin has been used to raise the skin in saddle-nose, to reinforce a poorly functioning sphincter ani, to make an artificial testicle, etc. (Gersuny, Eckstein, Stein).

Literature.—*W. Braun.* Klin.-histol. Untersuchungen über die Anheilung ungestielter Hautlappen. Beitr. z. klin. Chir., Bd. 25, 1899, p. 211.—*Brunner.* Wundinfektion u. Wundbehandlung II, Frauenfeld, 1898.—*Eckstein.* Hartparaffinprothesen. Berl. klin. Wochenschr., 1902, p. 315.—*Kredel.* Die angeborenen Nasenspalten und ihre Operation. Deutsche Zeitschrift für Chirurgie, Bd. 47, 1898, p. 237.—*v. Mangoldt.* Die Einpflanzung v. Rippenknorpel, etc. Chir.-Kongr. Verhandl., 1900, II, p. 460.—*Marchand.* Der Prozess der Wundheilung. Deutsche Chir., 1901.—*Payr.* Implantation der Schilddrüse in die Milz. Chir.-Kongr. Verhandl., 1906.—*Reinbach.* Untersuchungen menschlicher Granulationen. Zieglers Beitr. z. pathol. Anatomie, Bd. 30, 1901, p. 102.—*Ribbert.* Ueber Transplantation auf Individuen anderer Gattung. Verhandl. d. Deutsch. Pathol. Gesellsch. Zentralbl. f. allg. Pathol., Bd. 15, 1905. Ergänzungsheft, p. 104.—*Riggenbach.* Ueber den Keimgehalt accident. Wunden. Deutsche Zeitschr. f. Chir., Bd. 47, 1898, p. 33.—*Schloffer.* Ueber Wundsekrete und Bakterien bei der Heilung per primam. Arch. f. klin. Chir., Bd. 57, 1898, p. 322.—*Stein.* Paraffin-Injektionen, Theorie u. Praxis, Stuttgart. Enke, 1904.

II. ASEPTIC TECHNIC

All the methods employed in the treatment of wounds, before the nature and causes of wound infections were recognized, were unsuccessful. What were ill-directed attempts to determine the cause of and prevent wound infections, became definite and direct with Pasteur's discovery (1861) that fermentation and putrefaction of organic masses were caused by ferments of a vegetable or animal nature. Lister's suggestion (1867) that wound infections must have a similar cause has been most fruitful for the entire field of surgery, and remains to-day the most important milestone in its history.

The result of Lister's work was that an attempt was made to sterilize everything coming in contact with the wound, even the air. Lister used carbolic acid for this purpose, after it had been demonstrated that it would destroy the odor of sewerage and the intestinal worms which injured grazing cattle. It had, however, been used independently by an Italian surgeon since 1863. In the method as originally devised by Lister the skin, hands, instruments, sponges, sutures, and ligatures were sterilized with a five per cent solution of carbolic acid, and a spray of a two and a half per cent solution of carbolic acid was kept playing during the operation to prevent air infection, which was particularly feared.

The temporary and permanent dressings were also saturated with a solution of this acid. Lister's experiment was a success, and with one blow operative surgery was rid of its worst enemies—*hospital gangrene and the severe and frequently fatal putrefactive and pyogenic infections.*

In the antiseptic method of wound treatment, originally introduced by Lister, an attempt was made to prevent the development of wound infections and to combat those already developed by the use of different antiseptics. The year 1886 marks the beginning of the aseptic method of wound treatment, special emphasis being laid upon the prevention of wound infections, mechanical and physical methods of sterilization being chiefly relied upon. As in any innovation, a number of different complicated aseptic methods and procedures were introduced. Later investigations have shown that many of these are superfluous, and have compelled a return to simpler but as effective methods.

The science of bacteriology, which began with Koch's discovery of the anthrax bacillus (1876) and the introduction of solid culture media, required for making pure cultures of bacteria (1881), and was placed upon a firm basis by Rosenbach (1884), has shown how extensively the pathogenic bacteria are distributed. It is little wonder, after the brilliant confirmation of Pasteur's germ theory and the justification of Lister's suggestion that wound infections were caused by bacteria, that the latter's method as originally employed or modified by him was extensively used. In the early period of antisepsis the operating room resembled a carbolic acid bath. The carbolic acid spray was soon discarded in order to prevent infection through the air currents produced by its use. It was soon demonstrated, however, that the dangers of air infection were much less than those of contact infection through the hands, instruments, and dressings, and although the spray was discarded, antiseptic solutions, sometimes carbolic acid, at other times sublimate solution, which is active in much weaker solution, were still permitted to run over the wound during the course of the operation, the use of these antiseptics during the operation being considered very essential.

Later improved methods of investigation (Geppert) demonstrated that the bactericidal action of the antiseptic solutions in wounds and upon the surface of the body had been greatly overestimated. It was shown that antiseptics did not reach bacteria lying in the superficial epithelium and attached to foreign bodies; that they were inactive in wounds forming chemical union with the albuminous secretions; and that they destroyed the superficial bacteria only after long contact, injuring at the same time the tissues and viscera, thus reducing the natural resistance of the organism. In addition it was demonstrated that the antiseptic dressings, because of the volatility of the agents employed, had no marked bactericidal powers and even harbored bacteria (Schlange); that operation wounds treated with antiseptic solutions secreted more profusely and healed more slowly than those treated by the dry method (Landerer, von Bergmann), and that irrigation of severely inflamed tissues favored the extension of the infection (von Bergmann).

Guided by the results of bacteriological investigations of Koch, Gaffky, and Löffler (1881), surgeons turned to physical methods, of which sterilization by live steam and boiling water, excepting, of course, mechanical cleansing, are the most important. The entire procedure, to the perfection of which von Bergmann, von Esmarch, Landerer, Neuber, and Schimmelbusch have contributed most, is known as asepsis, and has replaced chemical sterilization by the use of antiseptics. Even at the present time antiseptics are indicated and required in certain cases, but they no longer as formerly are depended upon alone, being merely inci-

dental as contributing to the success of aseptic technic. It would be more correct to speak of physical and chemical antisepsis.

It would be impossible in a book of this character to discuss at length the different features of aseptic technic as employed by different surgeons, and besides it would be tiresome and confusing to the reader. Each method differs as to detail, but there is a general principle which is common to all. In the following chapters a simple but effective aseptic technic will be described. It can be easily followed by physicians and surgeons, who are often required to operate in private homes and do not have access to the conveniences of a hospital.

[A number of surgeons regard the use of head pieces, covering the hair and protecting the mouth and nose, as superfluous. Clinical experience, however, seems to indicate that the best results are obtained when the mouth and nose are covered either with a special mask or with sterile gauze and the head is covered with gauze or a cap. Rubber gloves are being very extensively used, being the best safeguard against infection of the patient, and at the same time protecting and preserving in good order the surgeon's hands. Rubber gloves can easily be prepared for any operation in private practice, and special masks for the head and face may be so easily procured or made when needed that they should be used in every case.]

CHAPTER I

PREPARATION OF THE SURFACE OF THE BODY

THE skin is the habitat of numerous varieties of bacteria, among which the ordinary pyogenic and putrefactive bacteria are most commonly found. The removal of these bacteria from the skin of the hands and the field of operation is a most difficult task, but one that is indispensable to successful results in surgery. The bacteria are hidden not only in the fat covering the skin, but also in the superficial layers of the epidermis, in the outer parts of the hair follicles, in the ducts of the sweat glands, and even in the most insignificant wounds and fissures of the epidermis. Haegler, after rubbing a culture of bacteria upon the skin, could demonstrate bacteria at all the points above mentioned, and they can be demonstrated in normal skin.

Sterilization of the Hands.—The results of the investigations of von Mikulicz, Haegler, Paul and Sarwey, Gottstein, and others have shown that it is impossible to completely sterilize the hands in a bacteriological sense for an entire operation. The surgeon and his assistants should

attempt to approach the ideal as closely as possible, and even when sterilization has been as thorough as possible, should regard the hands as very unreliable and exercise due precaution to prevent infections. The difficulties of hand sterilization may be easily recognized if the skin is examined under the lens, when all the fissures, which resemble the furrows in a newly plowed field, may be recognized, and one remembers that all the roughened areas, still more the small wounds and fissures, afford the best resting place for bacteria.

Of the different methods of hand sterilization which have been introduced, that of Fürbringer is the most extensively used, and is to be recommended. It is practiced in the von Bergmann clinic at the present time in the following way:

1. Mechanical cleansing of the hands and forearm for ten minutes in hot water with soap and brush; of the space beneath the free margin of the nail and nail folds with a nail file or cleaner.

2. Thorough drying of the hands and forearm with a sterile towel.

3. Washing for three minutes in from seventy to eighty per cent alcohol.

4. Washing for three minutes in 1 to 2,000 sublimate solution.

The first act is the most important. The hot water and soap and the vigorous rubbing with the brush remove the fats covering the skin, and loosen and separate the upper cornified layers of the epidermis.

[In the Bevan clinic the same methods are employed with two exceptions: (1) The bichloride has been entirely dispensed with, and (2) rubber gloves are invariably worn by the surgeon, assistants, and nurses.]

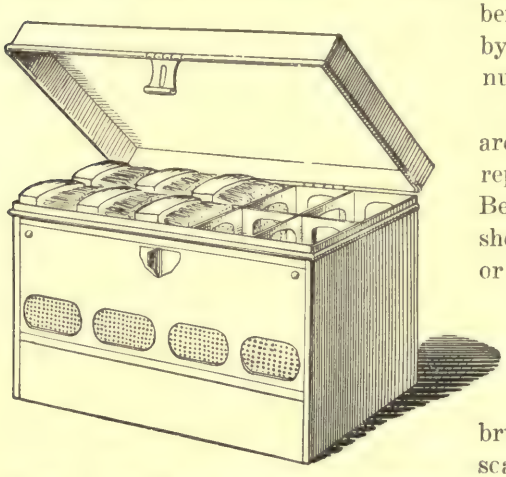


Fig. 42.—Lead Box with Trays for Brushes for Sterilization in Steam.

Simple bristle hand brushes are most valuable, and cannot be replaced by any other agent. Before using, a large number should be sterilized in a lead can or linen bag. In private practice it is recommended that the brushes be boiled in water and then kept in a 1 to 5,000 bichloride solution. The brushes should be freed of all soap before being placed in the solution, as soap forms a chemical compound with mercury, which is inactive. Five brushes will do very well for minor operations, and ten usually suffice for major ones.

After washing, each brush contains numerous bacteria, especially in its deeper parts. While the danger of transferring bacteria while washing under flowing water is probably not great, it has become a rule that a brush once used should not be used again until it has been sterilized. A number of brushes should be used in sterilizing hands to which grosser particles of dirt are attached, or which have been in contact with pus or fæces.

The dangerous space beneath the free margin of the nails and the nail folds demand especial attention. The grosser particles of dirt should be removed with a nail cleaner before washing is begun. Cleansing of the nails should be continued during the process of washing, as the finer particles to which the bacteria are attached become separated and may be removed with soap and brush. If the free margin of the nail is not longer than two millimeters, the space beneath can be cleaned very satisfactorily with the brush, as the bristles penetrate into all the depressions and recesses. The brush is far superior for this purpose to a number of substitutes such as orange-wood sticks, pumice stone, sand, different soap mixtures, etc., which have been introduced. Trimming the nails short has been recommended by some surgeons. It should be discouraged, however, as the space between the nail and the skin is then exposed, becomes rough and fissured, rendering sterilization more difficult.

Mechanical sterilization of the hands is an art to be acquired by practice, depending less upon the time actually spent in the process than upon the way and thoroughness with which the surface of the hands and forearm are washed and scrubbed. Haegler has recommended a method by which the thoroughness of the sterilization may be tested. After the fats are removed by washing, and the hands and forearms have been thoroughly dried, a few drops of liquid Chinese dye are rubbed into the skin. The surfaces are then scrubbed as usual with soap, water, and brush, and then examined with a lens. All the areas which have been neglected may be easily seen, as they will still be stained. One soon learns by these attempts how to wash his hands thoroughly. Special attention should always be paid to the space beneath the free margin of the nails, the deep furrows in the palm of the hand, the interdigital spaces, and the outer side of the forearm.

The water should be used as hot as possible. In all large institutions provided with running water the hot water may be regarded as germ free, in spite of the fact that it contains a number of harmless varieties of micro-organisms. Where there is no running water it may be boiled in kettles; after boiling for five minutes, it may be regarded as germ free.

The arrangements for washing vary. In clinical institutions and hospitals the bowls should be so constructed that they may be easily

cleaned, and that the water which is discharged from a tap may be turned on or off, and that the amount of hot and cold water may be regulated by the foot or forearm. [It is preferable that the stopcock be so arranged that the flow of water can be controlled by foot pressure. This is much more convenient, and there is much less danger of soiling the hands and forearm in an endeavor to turn the water off or to regulate the amount of hot and cold water discharged.]

In the simplest arrangement an attendant manipulates the stopcock, renewing the water when necessary, and regulating the amounts of hot and cold. In private practice an ordinary wash bowl which has previously been thoroughly cleaned with hot water may be used. In this case the water must be changed at least three times before the hands can be regarded as thoroughly sterilized.

All soaps used by the surgeon should be alkaline, as these favor the separation of the superficial layers of the epidermis. The tincture of green soap and soft soap are very extensively employed.

If in private practice one is compelled to use toilet soaps, mechanical sterilization must be more vigorous, in order to make up for the deficiencies of the soaps, which usually are fatty and neutral and do not favor the separation of the superficial layers of the epidermis.

After the hands and forearms have been washed and scrubbed thoroughly for the required length of time, they should be dried with a sterile hand towel. In this way the epidermis which has become loosened as a result of the washing is removed. The towel should not be used again until sterilized, as it takes up numerous bacteria.

Washing with Alcohol.—Washing with alcohol is a very important step in the procedure. When rubbed into the skin with sterile gauze or a brush it penetrates the deepest furrows, removing the fat and dehydrating the superficial layers of the epithelium, and prepares the skin for the action of the aqueous solution of the antiseptic which is used later. Alcohol, especially sixty to seventy per cent alcohol, has a certain sterilizing action. Alcohol of higher concentration has less bactericidal action, as it rapidly coagulates the albumen, forming a coating which prevents penetration. The skin already contains some water, remaining after the washing, and seventy or eighty per cent alcohol is therefore used.

After washing in alcohol the skin shrinks, and the small furrows and fissures in the epidermis in which the bacteria are lodged become closed. Cultures taken at this time would probably be sterile, but the hands should not be regarded as sterile in the surgical sense, for as soon as the hands come in contact with water or blood the fissures and furrows open and the bacteria are discharged upon the surface. For this reason the action of some antiseptic is required.

Bichloride of Mercury Solution.—Bichloride of mercury, introduced into surgical practice by von Bergmann (1878) and later recommended by Schede, is still at the present the most powerful chemical antiseptic.

A 1 to 2,000 solution of bichloride is employed, tablets prepared by any of the principal chemical manufacturers being used for the purpose. The addition of sodium chloride prevents the decomposition of the bichloride by the alkalies of the tap water. Warm tap water, boiled water, or water taken from a reservoir may be used in making the solution, as the bacteria which are contained in the water are killed by the bichloride after the solution stands for some time. The tablets used in making the solution contain a stain, and the solution is colored so that it can readily be distinguished from other antiseptic solutions or from water.

Other Methods of Hand Sterilization.—Brief mention will be made of other methods of hand sterilization. Some surgeons, among whom Neuber may be cited as an example, regard washing with hot water and soap as sufficient, while others, such as Ahlfeld, value the bactericidal action of alcohol so highly that they regard the use of other antiseptics as superfluous. Von Mikulicz attempted to combine the action of soap and alcohol by using a mixture consisting of 10.2 potassium soap, 0.8 unsaponified olive oil, 1.0 glycerin, 43.0 alcohol, and 45.0 water, which was rubbed into the skin with a brush. Haegler's investigations showed, however, that the bactericidal action of this mixture was only apparent, as it formed a thin layer of soap under which the bacteria were retained. It does not favor the separation of the upper layers of the epidermis to the extent that soap and alcohol do when used separately, and besides makes the hands slippery. Soap mixtures naturally prevent the bactericidal action of sublimate solutions, as the thin layer of soap, which remains attached to the skin, forms an insoluble, inactive compound with the mercury. In spite of these objections soap mixtures in solid form (Vollbrecht) or combined with pumice stone (Pfoerringer) may be used in case of emergency, where the water supply is low, especially in battle. It should be remembered, however, that they form but poor substitutes for the Fürbinger method.

Lysol is preferred by obstetricians. It is seldom used by surgeons, as it renders the hands slippery and interferes with the manipulation of instruments.

Cotton and Rubber Gloves.—The sterilized cotton gloves introduced by von Mikulicz, which were put on after the hands had been sterilized, have found but few friends. When used they must be changed from five to ten times during an operation, as the bacteria which come to the surface of the hands during the operation become attached to the inner

surface and may even be carried through to the outer surface. Von Bergmann, after a long and thorough trial, has discarded them for operative work, using them only when changing dressings, handling sterile sheets or towels and dry instruments.

Rubber gloves, recommended first by von Zoege-Manteufel, and perfected later by Friedrich, have a number of advantages. Rubber gloves may at first interfere with the dexterity of the operator and the delicacy of touch, but these disadvantages are soon overcome when the surgeon becomes accustomed to them. The hands should be sterilized before the gloves are put on. Gloves may also be boiled at the time the instruments are sterilized. They are then filled with sterile water and put on wet. Some surgeons prefer the dry method, the hands being covered with sterilized talcum powder before the gloves are put on, while a number of others prefer the wet method of using gloves. After they have been used, they should be washed off with soap and water, filled with gauze, and dried. Gloves should not be kept in antiseptic solutions, as they then soon lose their elasticity.

[Rubber gloves are being used very extensively by American surgeons. Bacteriology has demonstrated that hand sterilization, regardless of the method employed, cannot be entirely depended upon. Clinical experience has demonstrated the effectiveness of rubber gloves in preventing infections, and although it has frequently been demonstrated that the bacteria of the skin multiply beneath the rubber, they cannot reach the wound unless the glove is punctured and torn. Such accidents can be prevented by care, and after a little practice are recognized so soon that there is but slight danger of infection.

Gloves are especially valuable as a prophylactic measure, and should invariably be worn in making examinations of lesions which may be specific, and in examining or operating upon virulent infections. After gloves have been worn, the operator becomes accustomed to them, and they no longer interfere with the dexterity of the surgeon or his delicacy of touch. It is the belief of the editor that rubber gloves will be generally adopted, and that their introduction into surgery marks one of the greatest advances in aseptic technic.]

The Necessity of Washing the Hands in Sublimate Solution During the Operation.—The hands, unless chapped or fissured, may be regarded as comparatively free from germs after sterilization according to Fürbinger's technic, provided they have not been in contact with pus or other infectious material, as the most refined bacteriological methods fail to demonstrate any great number of bacteria in the skin. This condition does not persist, however, throughout an entire operation, even if the case is a clean one and the technic is good. Soon micro-organisms, especially the white staphylococci, appear upon the surface of the skin. The

researches of von Mikulicz, Haegler, Doederlin, and others have thrown light upon the origin of these bacteria. A few are derived from the air; the majority come from the deeper layers of the skin, from the outer parts of the ducts of the sebaceous and sweat glands, the hair follicles, and the small fissures in the epidermis. These bacteria hidden within these retreats have not been reached by either mechanical or chemical sterilization, and are carried to the surface by movements and friction, for example, in tying ligatures and sutures. Therefore the hands must frequently be washed in a sublimate solution, which should often be renewed during the course of the operation, and should come in contact with the wound as little as possible, tissue forceps and other instruments being used when possible (Koenig).

Care of the Hands.—The surgeon's hands should receive good care and be protected from infectious material. Prophylaxis is the best guarantee against infections. Therefore, rubber gloves should be worn when infected cases are dressed or operations performed upon suppurating or putrefactive processes. In examinations of the mouth or rectum gloves, or at least a finger cot, should be worn. After the operation is completed, the bichloride which remains attached to the epidermis should be removed with hot water and soap, as it may produce in susceptible people a vesicular eczema with secondary ulcers and crusts. After the last washing, when the hands have been thoroughly dried, glycerin or some hand lotion should be rubbed into the skin to prevent chapping. An infusion of bran has also been recommended for this purpose (Haegler). A surgeon whose hands are very rough should rub glycerin into the skin or apply lanolin before retiring, and wear gloves during the night.

Suppurating wounds of the hands, and even the most insignificant inflammatory processes make an aseptic operation impossible even if gloves are worn.

Sterilization of the Skin of the Field of Operation.—Each patient should be given a warm bath some time before the operation if there are no contraindications. Grosser particles of dirt (especially upon the hands and feet) should be removed by vigorous washing with ether, benzine, or petroleum ether. The field of operation and the surrounding skin should be shaved. The skin should be shaved even where there is but little hair, as the upper loosened epidermis is most effectively removed in this way. After the shaving is completed the same technic is employed as has already been described for hand sterilization.

Sterilization of Mucous Membranes.—Mucous membranes to be divided in the course of the operation can only be incompletely sterilized. Antiseptics have no effect upon bacteria contained in the secretion of

mucous membranes, and besides they may irritate the latter and be absorbed, causing severe toxic symptoms (e. g., bichloride poisoning after rectal and vaginal irrigations). Mechanical sterilization must be relied upon in these cases, a three per cent solution of hydrogen peroxide being used in the mouth cavity; sterile water or a bland, non-irritating solution (three per cent aluminum acetate, or two per cent boric acid solution) being employed for bladder and rectal irrigations. During operations upon the stomach and intestines the secretions of the mucous membranes, the stomach contents, and fæcal matter should be carefully wiped away with gauze sponges or laparotomy pads. The peritoneum should also be protected by laparotomy pads before the stomach or intestines are opened, and contamination of the peritoneum prevented.

Mercurial poisoning, the symptoms of which are salivation, colic, and persistent, often bloody diarrhœa, has not been observed after the use of 1 to 2,000 solutions. Haegler believes that surgeons who do not wash off the sublimate which becomes attached to the hands during sterilization may become slightly intoxicated by touching the lips with the fingers.

CHAPTER II

STERILIZATION OF INSTRUMENTS

Instruments are no longer sterilized by placing them in a two and a half per cent solution of carbolic acid shortly before or during an operation. At the present time they are sterilized by boiling, a rapid and efficient method. Pyogenic cocci are killed in a few seconds and the resistant anthrax spores in five minutes by boiling. Boiling for five minutes is sufficient in all cases.

Instruments which are sterilized frequently should be made entirely of steel without wood or horn handles, and should be thoroughly scrubbed before they are boiled. Those soiled during an operation should be rinsed off with cold water (preferably under the tap), and should then be allowed to remain for some time in a warm solution of soda and soft soap, scoured and well dried, and finally polished with alcohol and chamois skin. The more composite instruments (viz., artery clamps and forceps) should be taken apart each time for cleansing. Nickel plating of instruments is not necessary, and besides it is not permanent.

Soda Solution and Apparatus for Boiling Instruments.—To prevent the rusting of steel instruments Schimmelbusch has introduced the use of ordinary cooking soda (1 to 100). The addition of an alkali not only prevents the rusting of instruments, but also aids in sterilization, for the attached pieces of dirt are more easily separated and penetrated. A tablespoonful of soda is used in a liter of water. The apparatus de-

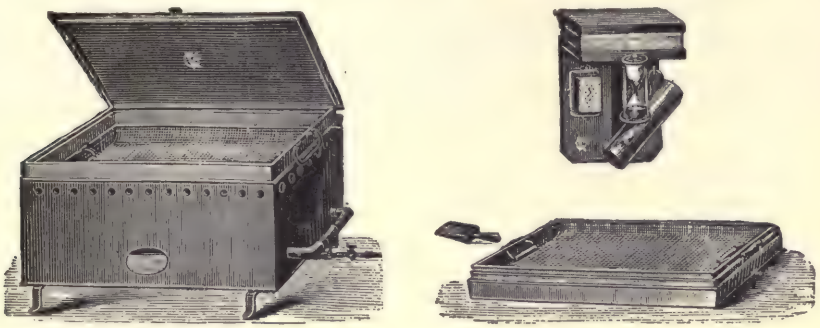


FIG. 43.—SCHIMMELBUSCH'S APPARATUS FOR STERILIZING INSTRUMENTS. It consists of three instrument trays, 14×18 inches, a graduate for making soda solution, wooden bracket for match safe, time glass and soda box. The apparatus is to be filled with water about two inches deep, to which a teaspoonful of soda is to be added. A large gas burner beneath sets the water boiling in a few minutes.

vised by Schimmelbusch, which is made in different forms and sizes, and differently equipped, is used for boiling. The one per cent soda solution which fills this boiler can be made to boil in a few minutes by an electric current, steam, gas, or spirit lamp. The instruments are placed in order in the flat, perforated tin tray, which is submerged in the solution. The edges of sharp instruments must be protected. For this reason needles should be placed in small glass or metal boxes, and the knives kept in a frame or the blades wrapped with cotton. The cover of the apparatus fits tightly and the temperature of the solution may be brought to 220° F. After boiling for five minutes or longer, the tray is removed by two steel hooks which are used for the purpose, and is placed in the frame of an instrument table. The instruments may be cooled by pouring cold sterile water over them, or by placing the tray in a basin of cold sterile water or spreading them upon a sterile towel; in the latter case some minutes will be required before they become cool.

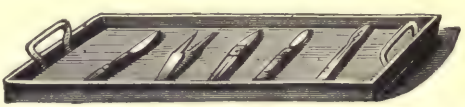


FIG. 44.—KNY-SCHEERER STERILIZING PAN WITH INSTRUMENTS FOR USE IN STEAM STERILIZER.

Instrument Table.—The instruments which will be required should be placed upon a table which is covered by a sterile towel; the other instruments being left on a tray or in a basin. The table should be so constructed that it can be easily placed near the surgeon or attached to an upright so that it may be swung over the patient. Instruments which have been used should be replaced by clean ones and should then be rinsed off, scoured, and resterilized. The soda solution should be kept in readiness. A spoon holding 10 c.c. may be used to measure the powdered soda or soda tablets may be used instead.

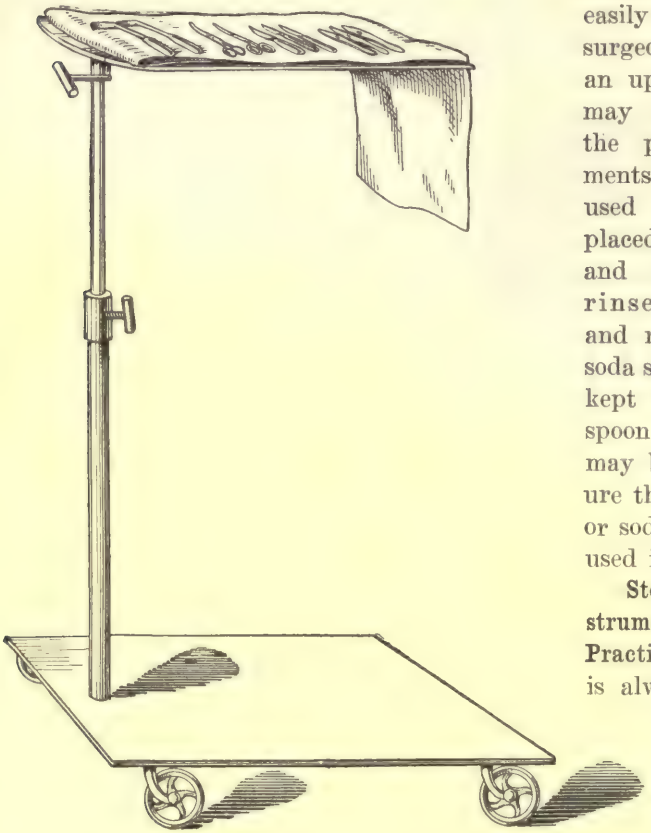


FIG. 45.—INSTRUMENT TABLE ON WHICH STERILE INSTRUMENTS REQUIRED FOR IMMEDIATE USE ARE PLACED. The table is provided with rollers so that it can be easily moved about and placed where convenient for the operator.

Sterilization of Instruments in Private Practice.—The surgeon is always able by the use of this soda solution to sterilize instruments rapidly and thoroughly in the patient's home. An ordinary kettle, if large enough, will suffice. If a large number of instruments must be sterilized, an asparagus boiler or a fish kettle with perforated tray may be used. The kettle is placed in cold water after boiling, and the soda solution cools rapidly, and the instruments may then be removed.

Instruments should not be washed or placed in sublimate solution, for they are soon blackened by a deposit of mercury. Syringes which are made of metal and glass and are provided with asbestos and glass pistons may be sterilized by boiling in the soda solution. To prevent

the glass from breaking the syringe should be half filled before it is placed in the solution, and it should never be placed directly in boiling water. The glass-rubber syringes cannot be thoroughly sterilized, and should not be used. Keeping them in antiseptic solutions is not sufficient, as the number of infections following their use in morphine injections demonstrates. Drainage tubes, silk, metal wire, and catheters may also be sterilized by boiling. Only the rubber and silver catheters, however, stand boiling. If the catheter has been used its surface must be rubbed off and its lumen cleaned by allowing tap water to run through it. The silk catheters covered with shellac and india rubber catheters should not be boiled, as they become soft and can no longer be used. According to Claudius they stand boiling best in concentrated salt solution (4 NaCl: 10 water), or according to Herman, in a concentrated solution of sulphate of ammonia (3:5 water), a procedure recommended by Elsberg for the sterilization of catgut. The ordinary Nélaton catheter, used so extensively in America, stands boiling very well, and may be sterilized with the instruments.

CHAPTER III

STERILIZATION OF SPONGES, BANDAGES, SHEETS, AND TOWELS; PREPARATION OF IODOFORM GAUZE

Gauze, cotton, and roller bandages are required in the dressing of the ordinary wounds.

Absorbent Gauze.—Absorbent gauze, which was introduced by Lister, is even to-day the most useful material for dressing wounds. It takes up wound secretion and at the same time allows it to dry. None of the substitutes which have been tried has this property. The substitutes are cheaper, but the absorbent gauze is indispensable. It is made of loosely woven cotton from which the fat has been extracted, and is sold in large bolts. These are cut into square pieces of about 25 qcm. with heavy scissors. Some of these are irregularly folded to form fluffed gauze, some are regularly folded to form dressings and compresses, or cut into long strips for tampons. A part of the fluffed gauze is used for sponges by which the blood is wiped away during the operation, while large amounts are used in dressing the wound.

Cotton.—Cotton packs easily when in contact with a wound, absorbs but little wound secretion, and forms a layer which prevents its discharge. It can never replace gauze, but it is soft and pliable and can be used to advantage in padding a bandage. It is made of bleached raw cotton

from which the fat has been extracted, and is sold in rolls. These are cut into strips about 15 cm. in width, which are rolled. Cotton is cleaner and more easily handled than the substitutes which have been introduced. The substitutes are, however, cheaper, and may be used for suppurating wounds which are discharging profusely. Wood cotton, made of wood wool and cotton, is rolled and used in the same way as cotton. Peat and moss,

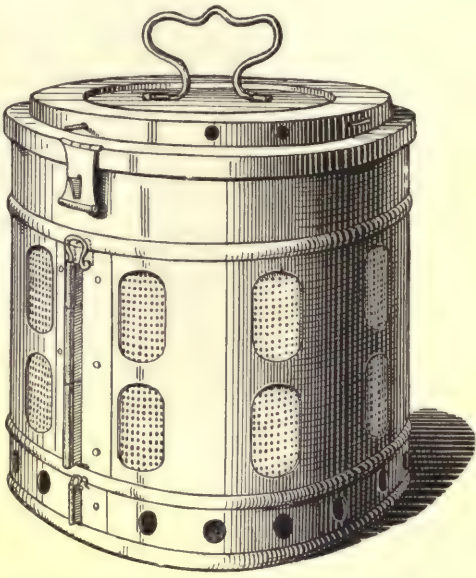


FIG. 46.—CAN FOR STERILIZATION OF DRESSINGS AND SPONGES. (After Schimmelbusch.)

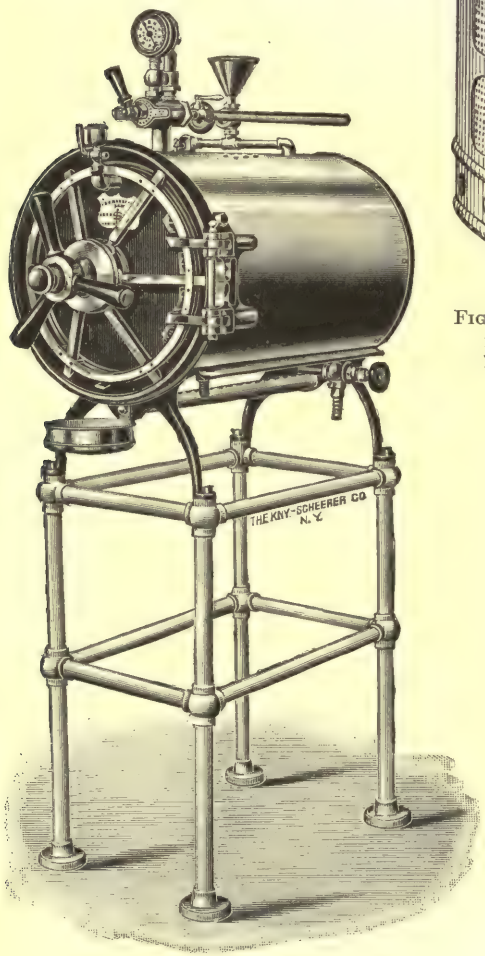


FIG. 47*a*.—PRESSURE STEAM DRESSING STERILIZER.

wood wool, cellulose, and other substitutes are sewed up in sterile bags and used for dressings in the form of pads. Moss felt and moss pasteboard are prepared from moss by running it through a press. They are covered with gauze before using, and are especially adapted for immobilizing dressings. After moistening, felt can be molded to the part to which it is to be applied.

Roller Bandages.—Muslin and gauze bandages are used for maintaining the dressings in position. The former, made of English mull of strong

fiber, are expensive but durable, and can be washed and used repeatedly. The gauze rollers, made of loosely woven German mull, are

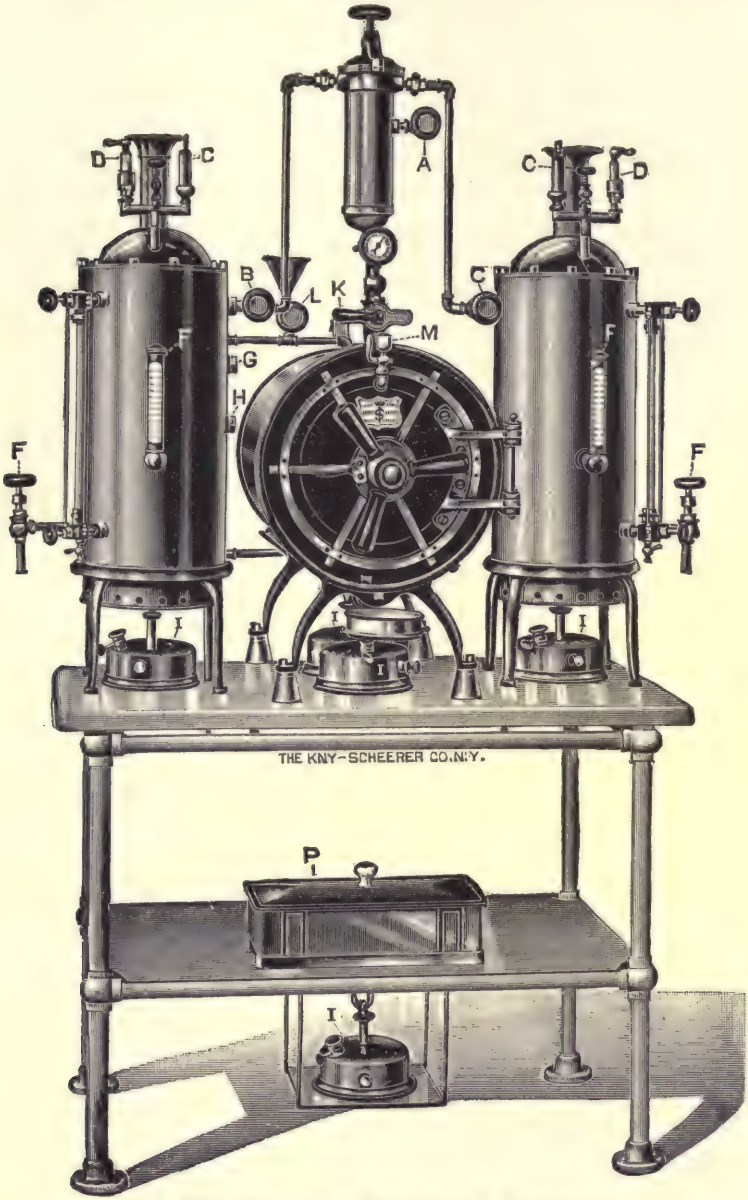


Fig. 47*b*.—Combination Sterilizing Apparatus.

cheap and pliable, but as a rule can be used but once. Roller bandages may be procured ready made from the dealers, or long pieces may be

torn or cut from the piece and rolled by machine. A few turns of starch bandage may be applied over the roller bandage to make it firm and prevent loosening. Starch bandages may be prepared by impregnating wide-meshed gauze with starch paste and allowing it to dry while on tension.

Sterilization of Dressings.—Materials used for dressings are sterilized by steam in an apparatus devised for the purpose. Tin cans of different forms and sizes are filled with gauze, cotton, and bandages, and are placed in the sterilizer. The can devised by Schimmelbusch is round or square. The cover, bottom, and sides of the cans are provided with a great number of holes which can be opened and closed at will by a strip of tin. These holes permit the steam to pass through the dressings. Sterilization is complete in three quarters of an hour after the steam begins to form (Schimmelbusch, Borchardt). After sterilization is completed the cans are allowed to remain in the sterilizer for a short time with the holes open in order to allow the materials to dry.

The cans are then closed and are ready for use. In hospitals freshly sterilized cans, some filled with gauze, some with cotton, and some with bandages, are supplied each morning to the dressing and to the operating rooms.

The sheets, the large and small towels which are used to bound the field of operation, to cover the patient, the operating table, and the small instrument table should be done up in packages and sterilized in the steam sterilizer. Moss, wood wool, and peat pads are handled in the same way.

Large hospitals are provided with a second large sterilizer which is used for the larger pieces, the smaller being sterilized in the apparatus which is used for the dressings. Some hospitals are provided with special steam sterilizers in which an entire bedstead may be sterilized by steam (viz., in epidemics).

Sea sponges, which were formerly extensively employed, are rarely used to-day to sponge wounds or as tampons (viz., laparotomies, resection of the maxilla). Aseptic gauze is used instead. Occasionally they are used upon artery forceps to wipe out the larynx and pharynx during anæsthesia, or with Beloque's tubes to tampon the nares in operations upon the nose and in nosebleed. Even here sterile gauze or rolled iodoform gauze may be used. Sea sponges do not stand physical sterilization well. Schimmelbusch has recommended that they be placed for one half hour in hot, but not boiling, one per cent soda solution for sterilization.

Preparation of Iodoform Gauze.—Iodoform gauze can be prepared in the following way: Long doubled strips of sterile gauze are spread out upon a sterile towel, the hands having been previously thoroughly

sterilized. The gauze is then powdered evenly with iodoform powder. An ordinary salt shaker which has been sterilized may be used for this purpose. The iodoform is then gently rubbed into the gauze with a sterile sponge, and ironed with a glass weight provided with a handle (Fig. 48). The strips of gauze are then rolled and are kept in a sterilized can. If the strips of gauze are sprinkled with a little sterile water, the iodoform powder becomes more easily attached. When the gauze is required for a tampon, the required length is pulled out from the roll with tissue forceps and cut with scissors. The can is then immediately closed. If the gauze contains too much iodoform, it can be shaken, and the greater part of the powder removed in this way.

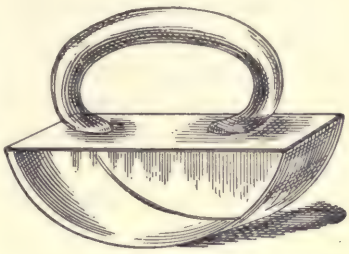


FIG. 48.

For minor cases (accidents) the gauze may be prepared, just before using, by sprinkling sterile gauze with iodoform powder.

The method above described is preferable to those methods in which the gauze is prepared with glycerin and colophony or sterilized in steam. In the first method the capillarity of the gauze is decreased, in the second the iodoform is decomposed. Besides, it is not necessary to sterilize iodoform gauze, for virulent pathogenic bacteria have never been found in iodoform powder, and clinically bad results do not follow the use of iodoform gauze which is not sterilized by heat just before being used.

The iodoform gauze sold by manufacturers is not to be recommended. It is difficult to handle, and furthermore the amount of iodoform contained in the gauze cannot be estimated, and the sterilization of the gauze cannot be depended upon. The gauze must be taken from the package with greatest care, to prevent contamination. It must be spread upon a sterile towel and the required length cut, and the remainder of the gauze must not be placed in the package again, but must be kept in a sterile can.

CHAPTER IV

STERILIZATION OF SUTURES AND LIGATURES

Silk.—Silk may be sterilized by boiling or in steam. It may be rolled upon glass or metal spools and boiled with the instruments, and then kept in an antiseptic solution. Schimmelbusch has devised for steam

sterilization small tin boxes which hold many spools of silk. These are closed when the sterilization is completed. When opened the end of the thread of each roll may be seen through a little opening in the side of the box.

Sterilization of silk by heat alone is not sufficient. Later investigations, particularly those of Haegler, have shown that in threading, tying, and passing sutures through the skin, bacteria become attached to the rough surface of the silk. These bacteria, while not very virulent, may develop in the silk and produce inflammation and suppuration about the stitch holes. If the suture is buried, a sinus may develop as the result of the inflammation, which continues to discharge until the suture is removed. For these reasons it is recommended that the heat and chemical methods be combined in the sterilization of silk. In the Kocher method the silk is treated for twelve hours with ether and alcohol to remove the fat. It is then boiled for ten minutes in a 1:1,000 solution of bichloride of mercury, and rolled upon sterilized glass spools after the hands have been thoroughly sterilized. These spools of silk are again boiled in a 1:1,000 solution of bichloride just before using. The albumen of the silk forms a chemical union with the mercury, which is slowly extracted by the juices of the body. The mercury gradually disappears from the suture in five to ten days, depending upon its size. These small amounts of mercury cannot destroy bacteria, but they check their development (Haegler).

Sublimate silk is especially adapted for buried sutures. In threading and handling suture material, it should be drawn through the fingers to remove the crinkles which are often present. Rough silk should be handled as little as possible in this way.

Silkworm Gut.—Silkworm gut is the fiber drawn out from the body of the silkworm killed just as it is ready to spin its cocoon. The surface of silkworm gut is smoother than that of silk, and therefore bacteria do not become attached to it so easily, and as it is more compact the bacteria do not penetrate into its interior. Silkworm gut is more expensive than silk and cannot be used for fine approximation sutures. It is used most extensively for tension sutures; silk or horsehair being used for the finer approximation of the edges of the skin. Silkworm gut may be sterilized by boiling with the instruments.

Metal Wire.—Metal wire is used especially in suturing bone and for buried tension sutures. Aluminum bronze wire is preferred, as it is ductile and durable. Silver wire is more rarely used. Wire can be sterilized with the instruments by boiling.

Catgut.—Catgut has the advantage of being absorbable. It is absorbed by the tissues within from two to four weeks, depending upon the size of the catgut. For this reason it is especially adapted for liga-

tures. The attempts to use catgut for sutures and ligatures date back to the time of Galen. Lister was the first to introduce it in a useful form.

Catgut is not prepared, as the name suggests, from the small intestine of the cat, but from that of the sheep. The mucosa, serosa, and greater part of the muscularis are scraped away, so that only the elastic submucosa remains.

After the gross cleaning, which is repeated several times, the whole intestine or long strips of it are twisted together like hemp rope. Rings of raw catgut, composed of strands from 3 to 5 m. in length, are sold by the dealers. The grade of the catgut varies a great deal.

Raw catgut contains all sorts of putrefactive bacteria and, besides, anthrax and tetanus bacilli. It must, therefore, be thoroughly sterilized before it is used. Catgut cannot be boiled in water, as it curls up and becomes brittle. Different methods of sterilization, which are partly chemical and partly physical, have been introduced; new methods are always being tried.

The method devised by Saul has been used in the von Bergmann clinic for eight years. The catgut is wound upon glass spools or slides, which are placed in eighty-five per cent alcohol (alcohol, 85; acid-carbolic liquefact., 5; aqua distil., 10). This solution is then slowly brought to the boiling point, which is about 168° F. (according to Saul between 172° and 176° F.). The spools or slides of catgut are allowed to remain in the boiling fluid from five to fifteen minutes, and then are either preserved in this solution or ninety per cent alcohol.

Of the other methods Hofmeister's may be mentioned. The catgut is allowed to remain twenty-four hours in a two to four per cent solution of formalin, and then is placed for twelve hours in water, after which it can be boiled in water from ten to thirty minutes, but its absorbability is decreased. The catgut is preserved in a sublimate-glycerin-alcohol solution (0.1 : 5.0 : 100.0).

A simple method, which has been tested for a number of years in the von Bergmann clinic, and which is to be recommended for practical use, is the following: The catgut is wound loosely upon glass rods or spools, which are placed in ether for twenty-four hours to remove the fat, the ether being kept in a sterile vessel. The ether is then poured off and the spools of catgut are placed in the following solution: Bichloride of mercury, 10; absolute alcohol, 800; distilled water, 200. This solution is renewed from two to three times in twenty-four hours, and is then replaced by ninety per cent alcohol, in which the spools of catgut are kept. If the catgut is too hard, glycerin (20 : 100) may be added.

In the Claudius method the catgut is wound upon glass slides or rolls, and is then placed in an aqueous iodin- potassium-iodid solution

(iodin 1, potassium iodid 1, water 100). This solution is prepared in the following way: The potassium iodid is first dissolved in a small amount of water, and then finely powdered iodin is added. The concentrated solution is then diluted until a one per cent solution is made. After remaining for eight days in this solution the catgut is ready for use. Shortly before the catgut is used it should be placed for a while in a three per cent carbolic or other sterile solution to remove the excess of iodin. Catgut not used during an operation should be replaced in the solution, which, according to Martina, should be changed each month. Catgut prepared by this method becomes black. It remains elastic, unless allowed to lie too long a time, when it becomes brittle.

Bartlett, of St. Louis, has devised a method for sterilizing catgut which has proved to be highly satisfactory. Catgut prepared by his method is used by a number of prominent American surgeons.

He describes the method as follows:

"The process can be divided into three stages: (1) The physical preparation of the material; (2) its sterilization; (3) its storage.

"1. The ordinary commercial ten-foot catgut strand is divided into four equal lengths, each of which is made into a little coil about one and a half inches in diameter. By twisting the last free end about four times around this little coil the latter will maintain its shape. These coils in any desired number (I usually make about a hundred and twenty at a time) are strung on a thread like beads, in order that the whole number may be handled at once. This string of coils is hung in a metal can—better still, in a beaker glass—but is not allowed to touch the bottom or sides. I suspend them by carrying the two ends of a thread through a small opening in a pasteboard cover which is placed on the receptacle. The same opening serves to admit a thermometer, which is carried down to exactly the point where the mercury bulb is on a level with the topmost coils. Liquid petrolatum is now poured in, the quantity being sufficient to immerse the catgut and the bulb of the thermometer.

"2. The vessel is set on a pan of sand, under which is placed a tiny gas flame of merely sufficient intensity to raise the temperature of the oil to 212° F. within from one to two hours. A little practice enables one to guess the size of flame necessary for this purpose. This is best done in the evening, and the temperature allowed to remain at about 212° F. (a few degrees' variation does not matter) until morning. The heat is then increased to such an extent that the temperature will run up to 300° F. in an hour; the gas is then turned off and the temperature of the oil allowed to return to 212°.

"3. The pasteboard cover, together with the string of catgut coils,

is lifted off, the superfluous oil is allowed to drop off, and then the thread is cut, allowing the coils to drop into the following mixture:

" Columbian spirits ..... 100 parts;
Iodin flakes ..... 1 part.

" The catgut is now ready for immediate use, and will keep without deteriorating for any length of time. The jar may be opened any number of times, so long as a sterile instrument is used for removing the coils, since the iodin protects the coils that are left behind from accidental contamination."—(*Surgery, Gynæcology, and Obstetrics*, August, 1906.)

Reindeer tendon sutures, which are absorbable, have been used (Greife). Kangaroo tendon has also been employed. Some surgeons (Kocher, Witzel, and others) have discarded catgut, and use sublimate silk for ligation.

CHAPTER V

OPERATING ROOM

Construction and Situation.—Every operating room should be so constructed that it may be thoroughly cleaned, should be well lighted, and so situated that it may be easily reached from all parts of the hospital. In order to meet the first requirement the walls, floor, and all the objects in the room must be made of materials which will not be injured by frequent washing with soap and washing soda. The walls should be made of tile, cement, glass or marble slabs, or should be enameled. The floor should be made of tile, cement, or other similar materials. The operating, instrument and bandage tables and the stands for basins should be made of enameled iron with wood, glass, or metal tops, and should be as simple as possible in construction. There should be no decorations on walls or ceiling, no corners or angles where dust might collect. The floor should be provided with a drain. Care should be exercised that grosser particles of dirt and highly infectious materials are not carried into the room where aseptic operations are performed.

Rooms Reserved for Clean and Infected Cases.—For this reason every large hospital has two operating rooms; one is reserved for clean cases, the other for infected cases. A suppurating wound should not even be dressed in a clean room. Larger hospitals have a large clinical amphitheater, a small operating room, in which such operations as a tracheotomy upon a patient with diphtheria might be performed, and special dressing rooms. In this way the aseptic operating room is protected

from contamination. Some surgeons demand that an adjacent room be used for washing, in order that they as well as the patient may be completely prepared before entering the operating room.

In clinical amphitheaters which are used for teaching purposes, it is impossible to divide the material into clean and suppurating cases. Quantities of dirt are always carried in by those attending the clinics, and the cleaning of the room must be more thoroughly done and the clean cases should be operated on first. Daily experience in a large clinic demonstrates that these precautions are sufficient.

Cleaning of the Operating Rooms.—The operating rooms should be cleaned daily after the operations are completed. The floors and the walls to the height of about 2 m. should be scoured with washing soda and green soap and rinsed off with water. A garden hose attached to a faucet may be used for this purpose. The operating, instrument, and bandage tables and stands for basins should be cleansed in a similar way. The enameled basins should be washed with soap and water and sterilized in a large steam sterilizer. The windows of the operating room should be left open for some time after each cleaning. The air is purified in this way.

Skylight and Arrangements for Artificial Light.—An operating room should be lighted by a skylight and wide side windows. If the ceiling is low and the windows high the skylight may be dispensed with. Usually the operating room is built so that it juts out, and thus three sides remain free and may be provided with large windows. As the glass is covered quickly with moisture and the water drips off, each window should be provided with a small drain. A double skylight is used to prevent the collection of moisture.

Electric light is preferred for artificial illumination. This is used in the form of a large portable incandescent light, provided with a reflector, which can be brought near the field of operation. A large number of incandescent lights arranged in a circle under a reflector is the most convenient form. If gas must be used, a number of burners which are provided with glass globes should be grouped under a shield.

Construction of Operating Tables.—Operating tables are differently constructed. They, likewise the instrument table and cabinets, must be so made that they may be easily cleaned, so that they will stand frequent scouring. Therefore only tables which are made of iron with glass, tin, metal, or wood tops should be used. The construction should be simple and strong. The table should be provided with a movable head piece, the middle piece so made that it may be elevated or depressed, the leg rests should be detachable, and provision should be made for the use of stirrups and for placing the patient in the Trendelenburg and other positions.

CHAPTER VI

THE ASEPTIC OPERATION

Preparation of the Patient.—The patient should be given a warm bath before the operation if his condition permits. This is the first step in the preparation of the patient, excepting, of course, those which are required to prepare for anæsthesia. He should be clothed in clean linen and placed upon an operating cart or in a freshly prepared bed, and taken to the anteroom of the operating room and anæsthetized. When the stage of excitement is passed the clothes should be removed and the patient covered with sterile towels and then lifted onto the operating table. This should be done slowly and carefully, the anæsthetist holding the head, an assistant taking the feet, and two strong assistants standing opposite placing corresponding hands upon the sacrum and lumbar region. In this way the heaviest patient may be lifted upon the operating table, and from the table to the bed. A broad rubber sheet, which should be changed before each operation should cover the table. A sterile sheet should be laid over this. The head roll which keeps the head in correct position during anæsthesia should be placed in a sterile pillow slip and placed under the neck or head as required. Sandbags and rolls, which are often required to secure the proper positions in many operations (e. g., kidney operations), should be covered with sterile towels or with sterile bags.

Sterilization of the Field of Operation.—A sterile assistant sterilizes the field of operation and the area surrounding it for some distance. In an operation upon the foot, the entire extremity; in an operation on the breast, the thorax, half of the back, and the arm on the same side should be sterilized. Wounds, fistulæ, ulcers, or ulcerated tumors which are present in the field of operation should be covered with sterile gauze while the surrounding area is sterilized. In this way the carrying of infection to the adjacent tissues will be prevented. Suppurating ulcers should be covered with iodoform gauze and adhesive plaster or thoroughly cauterized with a Paquelin cautery.

When the cleansing is completed, the patient is elevated and the wet towels are removed. The rubber sheet and the patient's back are dried, and a warm sterile sheet which covers the entire table and the head roll is placed under him. It is best to prepare the patient in the anteroom upon an operating cart, and when fully prepared he can be lifted onto the operating table. Sterile, warm towels are then spread over the patient and the hair is covered with a hand towel. Only the face and the field of operation to the extent of the proposed incision should be exposed.

The towels last applied should be fastened together by safety pins or artery forceps, being used in this way in preference to the ordinary laparotomy towels. Towels soiled during the operation should be replaced by clean ones. Packages of clean sterile towels should be placed upon the top of the sterilizer. They become warm, and when applied prevent the patient from becoming cool, and render the use of a table which may be heated superfluous.

The anæsthetist should hold a sterile towel in front of the patient's face to protect the wound from the patient's breath, particularly from expectoration, vomitus, and particles expelled by coughing.

Preparation of Surgeon and Assistants, and Arrangement of Instruments, Sponges, etc.—All those taking part in the operation should put on sterile linen gowns after their hands are sterilized. Rubber aprons may be worn under the gowns to protect the clothing. Finger rings should, of course, be removed.

The instruments and dressings should be placed near the operator. A trained nurse, who has been trusted with the preparation of the instruments, arranges them in order upon a table which is covered by a sterile towel. She replaces the instruments used during the operation by fresh ones, hands instruments, sutures, and ligatures. The sponge box should be placed near the surgeon, so that he can help himself to instruments and sponges as much as the character of the operation permits. Basins filled with sublimate solution should be placed near him, so that he can wash his hands repeatedly during the operation.

The instruments and sponges should be handled as little as possible. Only the operator should put his hand in the wound, and he only in case it cannot be avoided (laparotomy, etc.). Many manipulations may be performed with tissue forceps and sterile gauze. For this reason many surgeons allow their assistants merely to hold retractors or to hold and steady tissues (flaps in plastic operations, intestinal loops) with layers of gauze. As a rule, the operator will be able to apply artery forceps and ligatures. We know that it is impossible to keep the hands sterile during the progress of an operation, and it should be an invariable rule to wash the hands and the skin adjacent to the incision frequently with sublimate solution or sterile water in order to remove the blood in which the bacteria lodge.

Air Infection.—Besides the contact infection, which may be prevented by thoroughly sterilizing the hands and observing other precautions, air infection must be considered. Air infection is usually not to be feared, for the air of a thoroughly cleaned operating room contains but few pathogenic bacteria (Schimmelbusch, Symmes). The wound may become infected if the room is dusty or if drops of fluid from the patient's or surgeon's mouth gain access to it. If the surgeon coughs, sneezes, or

speaks, small drops of fluid may be carried into the wound, and he should therefore exercise due precautions. Experience has demonstrated that air infection may be prevented if the arrangements in the operating room are correct and due precautions are taken. The operating room should not be cleaned just before an operation, and it is not necessary between each operation. Soiled sponges and linen should not be thrown upon the floor, but in pails or basins, and dressings should not be changed just before or during an operation. Anyone with a nasal or pharyngeal catarrh should remain at some distance from the operation, and in speaking, sneezing, or coughing, the head should be turned away from the field of operation. A towel should be held in front of the patient's mouth and there should be no draughts. [The mouth masks introduced by von Mikulicz, and the gauze veils preferred by other operators, are of great importance and should be worn by the surgeon and his assistants.]

The wound should be kept covered with gauze as much as possible. This is the best protection against infection, and besides it controls capillary hæmorrhage and prevents the surface of the wound from the harmful effects of drying. Some surgeons prefer compresses which have been moistened with warm physiological salt solution.

The more rapidly the operation is performed, the less the danger of hand and air infection. For this reason an operation should not be delayed by needless conversation and indecision. In this way the time of anæsthesia is lessened and the general condition improved. A superficial showy operation should, however, never be performed at the expense of asepsis and thoroughness.

Schloffer and Brunner and others have demonstrated that even in wounds which heal by primary intention, large numbers of bacteria may be found a few hours after operation. These bacteria come especially from the skin of the hands, and fortunately are rarely pathogenic and virulent. It is difficult to predict how these bacteria will act, therefore it is a rule that the surgeon should not come in contact with pus, fæces, and highly infectious material. For this reason all examinations, operations, and change of dressings, in which this is unavoidable, should be made with rubber gloves. Virulent bacteria, once having invaded the furrows and fissures of the skin, are as difficult to remove as the harmless bacteria ordinarily found there. These bacteria may reach the surface during the operation and be transplanted into the wound. Therefore it has become a rule, which is based upon clinical experience, never to open a phlegmon just before an aseptic operation.

The Necessity of Avoiding Rough Manipulations, Lacerations, or Crushing of the Tissues.—The bacteria introduced into the wound during an operation usually do not interfere with primary healing, as they are attenuated and are destroyed by the bactericidal properties of the

tissues. If the tissues are roughly handled, torn, or crushed during the operation these bacteria may develop and cause inflammation. Accumulations of blood and wound secretion also favor their development.

Incisions should therefore be clean cut, and blunt dissections with tissue forceps and gauze should be made only in loose tissues. Thorough control of hæmorrhage and rational wound treatment counteract the growth of bacteria. The more one is compelled to handle wound surfaces, the longer they are exposed, and the more they are contused, the greater the possibilities of infection. If it is probable that there will be a large amount of wound secretion, provision should be made for its escape, and for this purpose spaces left between the sutures and deep cavities should be drained and tamponed.

The Duties of the Operator.—The operator is not only responsible for the operative work, but superintends, as far as he is able, the asepsis and the administration of the anæsthetic. In order that he may do this, the discipline of his help must be perfect, and all his wants must be immediately and correctly attended to. For this reason most surgeons prefer to operate with their own assistants and nurses.

Closure of the Wound.—An aseptic operation-wound is closed by sutures or by a plastic operation after the hæmorrhage has been controlled. Wherever it is probable that there will be a large amount of wound secretion, its accumulation should be prevented by leaving spaces between the sutures, in which should be inserted strips of iodoform gauze or gutta percha. This is necessary particularly after the division of a large number of lymphatics (axillary fossa, inguinal region, neck), and where hæmorrhage cannot be perfectly controlled, as in operating upon a tumor. A tubular drain should be inserted in deep cavities, which experience teaches secrete profusely, as is the case after the axillary fossa is cleaned out. There is no danger that bacteria will develop along the tube, for they do not pass against the current of wound secretion. Compression of the wound by bandages prevents the accumulation of wound secretion, and a graduated gauze compress exerting gentle pressure should be laid over the deep, sutured wound, such as is made in the extirpation of a tumor, and held in place by adhesive straps.

The Dressing of Aseptic Operation Wounds.—As a rule, sutured operation-wounds are covered with a sufficient number of sterile dressings, over which cotton is laid. The dressings are so arranged that evaporation of the wound secretion will not be interfered with. The gauze should dry the wound secretion to prevent the development of bacteria and decomposition. Bony prominences should be covered with cotton, and a roller bandage applied evenly, but with not too great pressure. On the extremities the roller bandage should always be applied from the periphery toward the trunk. A few turns of a moist starch bandage may

be applied over the roller to make it firm. As any movement may do harm to operation-wounds, immobilizing papier maché, wood, or tin splints well padded with cotton are often used.

An anæsthetized patient may be carefully held in a half-sitting posture while a bandage is applied to the thorax. In applying bandages, to the pelvis or abdomen a pelvic support, made of metal so that it may be sterilized, is frequently used.

Immediate After Care of the Patient.—After the bandage has been applied the patient should be carefully placed in a warm bed and a warm, dry shirt put on. The operator should see that the position in bed is correct, the head being but slightly elevated. If the extremities have been operated upon they should be elevated and supported by pillows or sandbags, and movement should be prevented by the use of sandbags placed on either side. The bedclothes and shirt should be smooth, particularly about the coccyx and sacrum, for wrinkles may rapidly produce decubitis. A rubber cushion filled with water should be placed under the sacrum of old and emaciated patients. It is agreeable to many patients to have a pillow or roll placed under the knees (especially after abdominal operations). Many patients who have been able to be about before the operation, complain of severe lumbar pain, which often lasts for a week. This pain is often controlled by placing a soft roll under the sacrum; frequently morphine is required.

The pain in the wound usually disappears after one half hour, and if the patient does not recover from the anæsthetic rapidly it is scarcely perceptible.

Most patients do not sleep the night following the operation. This is due to the discomfort caused by the quiet position, the excitement of the operation, and the after effects of the anæsthetic rather than to the pain. [Narcotics, such as morphine, should be dispensed with in the after-treatment if possible.]

The excitement of the patient, which results partly from the operation, partly from the anæsthetic, is best controlled by quiet and rest. As soon as the patient is awake a short and comforting statement should be made concerning the operation and the prospects of a rapid convalescence. It is the custom of many surgeons to discourse at length upon the condition of the patient and to give a correct prognosis. This should not be done, as it disquiets the patient and often the statements of the surgeon are misunderstood.

During the after-treatment the wants of the patient should be attended to and his confidence retained.

The nourishment during the first few days should be strengthening and nutritious, but the after effects of the anæsthetic should be kept in mind. Special orders for feeding should be given after operations

upon the stomach. Alcoholic drinks should only be prescribed when the patient is a drinker. Patients may be allowed to smoke if accustomed to it, provided there are no diseases of the respiratory tract.

Increasing pain in the wound and an elevation of temperature indicate some disturbance in wound healing. Severe infections begin with a chill; the temperature reaches 102° F. and is higher instead of lower on the following day. If this happens the dressings should be immediately changed, some sutures removed, and a light dressing applied without pressure. If the temperature does not subside more sutures should be removed and the wound tamponed and drained.

Mild inflammation of stitch holes is indicated after a few days by a slight elevation of temperature and some pain.

Frequently the evening temperature on the day following the operation will reach 101° F. If it is higher on the second day, the probabilities are that the wound is infected; if lower, it is probable that the temperature will soon return to the normal and that healing will occur by primary union. Fever, which is never accompanied by general symptoms and soon subsides, frequently develops at the end of the first or the beginning of the second day following the operation. The cause of this fever is not known. It has been suggested that it follows the absorption of fibrin ferment, and has been called *ferment*, *aseptic*, or *noninfectious* fever. It will be discussed more fully in the chapter devoted to the general discussion of fever (p. 167).

Pain in the wound and slight elevation of temperature without general symptoms are frequently caused by an accumulation of blood. The dangers which follow infection of hæmatomas often deter the surgeon from opening the wound and removing the clots. If a large hæmatoma develops and it is probable that large vessels are bleeding, the wound should be opened widely, the clots removed, the vessels caught and ligated. Asepsis should be as perfect as in an operation. After the hæmorrhage is controlled, the wound should be resutured, the sutures being placed at wide intervals, and drainage with iodoform gauze should be maintained for some days.

If the clinical course of a sutured wound is undisturbed the dressings may be removed, as a rule, in from one week to ten days. If the wound is tamponed or drained the dressings should be changed after five days or earlier. After the removal of sutures, drainage tubes, and tampons the edges of small wounds may be drawn together with gauze and adhesive plaster. If the wound is large, immobilization and compression may be required for a longer period.

Dressings should be changed upon an operating cart or table, if the patient's condition permits, and not in bed. While the dressings are being changed, the bed should be freshly made.

CHAPTER VII

ASEPSIS AND THE ASEPTIC OPERATION IN PRIVATE PRACTICE

THE aseptic technic of the surgeon who is compelled to perform minor and emergency operations, such as strangulated hernias, tracheotomies, appendectomies, and amputations for crushing injuries, and to care for wounds in patients' homes must be simple, yet effective. A certain equipment is required as the foundation of his surgical activity, such as:

1 apparatus in which both instruments and dressings may be sterilized (Fig. 47*a*, p. 64), or
1 small steam sterilizer and
1 can for dressings (Figs. 43, 46) and
1 instrument boiler.
3 gowns.
6 to 12 rough hand towels made of linen.
1 instrument case in which to keep instruments.
10 bristle brushes.

Gauze and cotton may be bought in large packages of the dealer direct, and cut into required sizes and lengths when needed. One or two rolls of sterilized gauze and a roll of iodoform gauze should always be included.

Gauze and starch bandages should be bought of the dealers. Druggists ask a very high price for these, and it is much cheaper to buy them from the manufacturer direct.

Rubber drainage tubes should be boiled with the instruments and fenestrated with curved scissors just before being used.

Sufficient amounts of silk and catgut should always be kept in readiness. Silk should be wound on glass slides, catgut upon glass spools. The former should be boiled and preserved in an alcoholic solution of sublimate; the latter after special preparation should be preserved in an alcoholic sublimate or iodin solution. The jars in which the silk and catgut are preserved should be provided with closely fitting tops.

Rubber catheters should be sterilized by boiling just before they are used.

The surgeon may prepare his own plaster of Paris bandages, or buy them of the dealers or druggists. In preparing these a dry starch bandage is spread out upon a board and plaster of Paris is sprinkled over it and rubbed in evenly with the edge of a ruler. The bandages are then loosely rolled and placed in tin cans to protect them from moisture.

Papier maché splints, made by papier maché manufacturers, 6 cm. broad and 1 m. long, may be used for immobilizing dressings. These splints may be molded to fit, and are more practical than the wood and tin splints, which rarely fit.

The things required for an operation should be prepared and packed in the following way:

(*a*) For a minor operation (felon, incision of a boil, excision of an atheroma, etc.), the following instruments should be boiled:

2 knives.
1 rat-tooth forcep.
1 anatomical forcep.
2 sharp hooks.
2 artery forceps.
1 pair of scissors.
1 syringe for local anæsthesia, if the operation is not to be performed with æthyl chloride.
2 brushes.
Iodoform gauze.
Sterile gauze.
1 roller bandage.
1 roll of cotton and sublimate tablets are then added.

The instruments are laid upon a sterile hand towel with sterile forceps and wrapped in it. The hand towel, which contains everything needed, is wrapped in strong paper or better is placed in a canvas bag (*vide* Fig. 49).

(*b*) In case of major operations (herniotomy, sequestrotomy), it is well for the surgeon to mentally review the instruments, so that none will be forgotten.

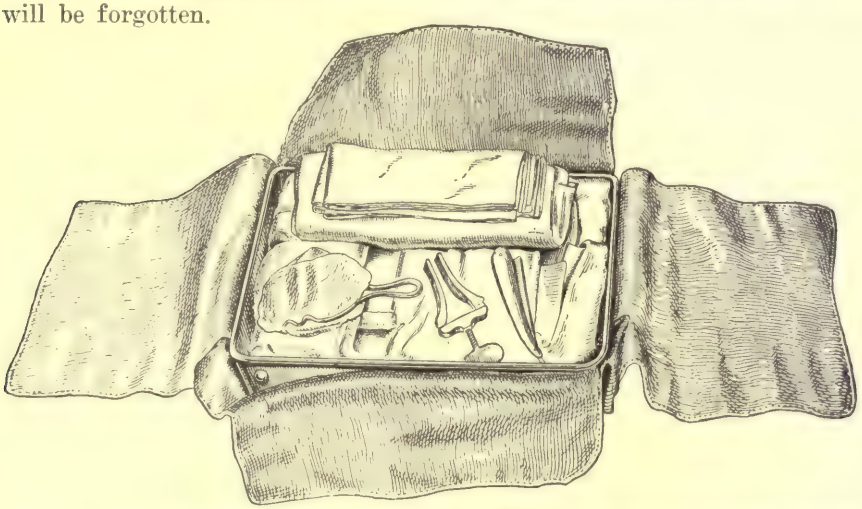


Fig. 49.—Canvas Bag for Carrying Supplies Which May Be Used in Private Practice. Bag open.

The following are necessary for operations upon soft parts:

3 knives (covered with cotton or lying in a frame).
2 anatomical forceps.
2 rat-tooth forceps.
1 pair of straight scissors.
1 pair of curved scissors.
10 artery forceps.
1 grooved director.
2 sharp hooks,
2 blunt hooks.
1 aneurysm needle.
1 box filled with needles.
1 needle holder.
1 nail cleaner.
Drainage tubes of different sizes.
1 syringe (for local anæsthesia, morphine, or camphorated oil).

The following should be added for a tracheotomy:

2 silver canulæ of different sizes provided with tapes.
1 sharp hook.
1 Bosescher retractor.

Or for an operation upon bone:

3 bone knives.
1 periosteal elevator.
2 chisels.
1 mallet.
1 Esmarch constrictor or rubber tubing.
1 sharp spoon.
1 Luer bone-cutting forcep.
1 bone scissors.
5 wire saws.
1 keyhole saw.

For bone suture:

1 drill and aluminum-bronze wire.

For amputation:

1 amputation knife.
1 amputation saw (or only wire saws).

FIG. 50.—BAG CLOSED.

When the instruments are thoroughly sterilized the tray is taken out of the instrument boiler and is placed upon a large sterilized towel. The tray is then wrapped in the towel and the following are laid on top:

Things required for anæsthesia—

1 roll of iodoform gauze wrapped in a sterile hand towel.
6 pairs of rubber gloves wrapped in gauze.
1 bag containing 6 bristle brushes.
1 razor.
1 bottle of sublimate tablets.
1 jar of silk.
1 jar of catgut.
2 bars of good alkaline soap.
1 bottle of cocaine tablets.
1 bottle of morphine tablets and camphorated oil.
1 bottle of 70–80 per cent alcohol (250 c.c.).
Some rolls of gauze and roller bandages, which are wrapped in a sterile towel.
2 operating gowns.
Operating and hand towels should also be added.

The package is then placed in a tin-lined canvas bag, which may be easily sterilized in a steam sterilizer. The canvas bag is fastened by straps.

One or two packages of gauze, depending upon their size, are put in another canvas bag.

Another bag is often required for the sheets and linen used in major operations.

Plaster of Paris bandages (also alum powder) should be carried in tin boxes in which they are kept.

Papier-maché splints may be carried under the straps of one of the canvas bags.

If a number of operations are to be performed, it is well to take an instrument boiler along.

When an operation is to be performed in a house, that room should be selected which has the best light and is used the least, as the danger of air infection is thus reduced. The conditions in a sleeping room are unfavorable, for the air is contaminated with dust and, besides, it is difficult, very often impossible, to perform operations in bed. Only the most insignificant operations should be attempted in this way.

Only those things should be removed which interfere with the operation. The taking down of pictures and curtains, cleaning the floor, etc., raises so much dust that at least a day should intervene before the operation is performed.

An extension table (or two small tables may be placed end to end) should be placed near a window or under a chandelier, and covered with several blankets, the head of which is elevated by a cushion or roll. A rubber sheet should be placed over the blankets to protect them from the wet. A large, fresh sheet should be spread over the rubber sheet, and the improvised operating table is then complete. While the operating table is being prepared, water should be boiled in a number of large kettles. Four or, better, six washbowls should be cleaned with green soap and rinsed with hot water.

A good-sized table (a sewing table answers the purpose very well) covered with a sterile towel should be placed at the side of the table where the operator stands. After the hands have been sterilized, the contents of the tray above described should be emptied and arranged upon this table. The surgeon is then able to help himself to instruments, gauze, sutures, and ligatures, all of which should be covered with a sterile towel until required.

Freshly washed linen sheets, tablecloths, and hand towels may be used for a number of different purposes. If time permits, they should be ironed with a hot iron just before being used. In this way the sterile sheets and towels may be saved and used for sterilizing the hands and drying and protecting the field of operation.

Washbowls containing alcohol, sublimate solution, and hot water should be made ready. Water cans and pitchers filled with sterile water should be kept in readiness.

An attendant, a nurse, and a physician are required in an operation performed without general anæsthesia. When an anæsthetic is given the surgeon should have another colleague or an assistant, who merely

holds the retractors placed by the operator or hands instruments, a nurse, and an attendant to pour water, prepare sublimate solution, and hold basins.

All the rules of asepsis should be rigidly observed. Long exposure of wound surfaces and touching them with the fingers are followed by infection more frequently than is the case when the operation is performed in a regular operating room. For this reason the operation should be performed rapidly but thoroughly, and rubber gloves should always be worn. It should also be borne in mind that the conditions for caring for the wound are unfavorable; therefore the sutures should be placed far apart, and whenever the tissues have been lacerated or have been touched by the fingers, an iodoform tampon or gutta percha drain should be inserted.

It may be readily understood why only a limited number of operations can be performed with safety outside of the hospital. The only aseptic operations which should be undertaken are those which may be rapidly performed. Operations upon inflammatory processes are particularly adapted for this work. Long and difficult operations should be performed in the patient's home only when absolutely necessary (e. g. amputation of thigh, disarticulation for crushing injury, laparotomy for ileus). [With a complete emergency outfit and well-trained assistants, almost as satisfactory aseptic work can be done in a private house as in a hospital.]

All the instruments and dressings required for any emergency operation may be easily carried in two medium-sized traveling bags. The Bevan emergency bag contains the following:

1 emergency bag containing—
1 amputating knife for hip and thigh.
1 amputating knife for leg and arm.
6 minor operating knives.
1 Emmett's uterine needle holder.
12 Tait's artery forceps.
1 pair Collin's retractors.
2 plated probes.
2 grooved directors.
1 pair straight scissors.
1 pair curved scissors.
1 pair strong scissors, 6 inches.
1 pair Liston's bone forceps.
1 pair curved lithotomy forceps.
1 bone curette.
1 set Brainard's bone drills.
1 thermometer.
1 metal case for 2 amputating knives.
1 metal case for 8 smaller knives.
1 razor in case.

1 dozen assorted uterine needles.
1 dozen assorted surgeons' needles.
1 hypodermic syringe and tablets in an aluminum case.
1 set of Murphy's buttons.
1 Knight's stethoscope.
1 razor strop.
1 4-oz. metal-cased bottle for alcohol.
½ pound of ether.
1 bottle of sublimate tablets.
1 coil silkworm gut.
1 hard rubber iodoform duster.
1 16-in. telescope case containing:
6 plaster of Paris bandages (3 in. by 6 yards).
1 can of sublimate gauze.
1 can borated gauze.
½ pound of absorbent cotton.
½ dozen gauze bandages (2 in. by 10 yards).

½ dozen cotton bandages (2 in. by 8 yards).
2 bottles of assorted catgut.
1 bottle kangaroo tendons.
1 Bouchard's syphon syringe.
1½-ounce hard rubber syringe.
1 papier maché catheter case.
6 dressing splints.
2 plated male catheters.
1 Buck's lithotomy staff.
1 Rongeur forceps with spring.
1 pair Ferguson's bone-holding forceps.
1 lead mallet.
1 Van Buren's bone chisel.
1 Van Buren's bone gouge.
1 mastoid chisel.
1 Galt's trephine.
1 metacarpal saw (movable).
1 Kocher's director.
1 Sayre's periosteotome.
2 aneurysm needles (metal handled).
3 hard rubber trachea tubes.
2 mouse tooth forceps (spring).
1 Volkmann's curette.
2 Nélaton's catheters (soft rubber).
3 filiform bougies.
2 Hunter's wedges.
1 bottle of assorted drainage tubes.
1 Esmarch's bandage.
1 8-oz. metal-cased bottle for chloroform.
1 Esmarch's chloroform inhaler.
3 duck rolls for instruments.
1 Esmarch's tongue forceps.
½ dozen flannel bandages (4 in. by 8 yards).
2 brass pulleys.
3 dozen safety pins.
1 pure rubber bandage (2½-in.).
2 rolls adhesive plaster (1 and 2½-in.).
6 sterilized laparotomy pads.
8 sterilized towels.
1 can of iodoform gauze (10 per cent).
20 tubes of sterilized silk.
Needles for intestinal sutures.
2 aprons.
1 nail brush.

LITERATURE.—*v. Bergmann.* Zur Sublimatfrage. Therap. Monatshefte, February, 1887, p. 41.—*Borchardt.* Die Desinfektion unserer Verbandstoffe. Archiv f. klinische Chirurgie, Bd. 65, 1902, p. 516.—*Brunner.* Weitere Versuche über Katgutsterilisation. Beitr. z. klin. Chirurgie, Bd. 7, 1891, p. 447.—*Claudius.* Eine neue Methode zur Sterilisation der Seidenkatheter. Zentralblatt für Chirurgie, 1902, p. 465;—Eine Methode zur Sterilisierung und zur sterilen Aufhebung des Katgut. Deutsche Zeitschrift für Chirurgie, Bd. 64, 1902, p. 489;—Erfahrungen über Jodkatgut. Ebenda, Bd. 69, 1903, p. 462.—*Cleves-Symmes.* Untersuchungen über die aus der Luft sich absetzenden Keime. Arbeiten aus der v. Bergmannschen Klinik, Part 6, 1892.—*Elsberg.* Ein neues und einfaches Verfahren der Katgutsterilisation. Zentralbl. für Chir., 1900, p. 537.—*Friedrich.* Das Verhältnis der experimentellen Bakteriologie zur Chirurgie. Leipzig, 1897.—*Greife.* Renntiersehnenfäden als Naht- und Ligaturmaterial an Stelle des Katguts. Münch. med. Wochenschr., 1901, p. 1005.—*Haegler.* Händereinigung. Basel, 1900;—Ueber Ligatureiterungen. Chir.-Kongr. Verhandl., 1901, II, p. 258;—Wundverbandmittel in Kochers Enzyklopädie.—*Herman.* Ueber das Sterilisieren der Seidenkatheter. Zentralbl. f. Chir., 1901, p. 63.—*Hofmeister.* Ueber Katgutsterilisation. Beitr. z. klin. Chir., Bd. 15 and 16, 1896.—*Koch.* Untersuchungen über die Aetiologie der Wundinfektionskrankheiten, 1878.—*Kocher.* Chirurgische Operationslehre. Jena, 1902.—*König.* Aseptik der Hände? Operationen ohne direkte Berührung der Wunde mit Finger u. Hand. Zentralbl. f. Chir., 1900, No. 36.—*Fritz König.* Das neue Operationshaus in Altona. Archiv für klin. Chirurgie, Bd. 70, 1903, p. 1078.—*Martina.* Die Katgutsterilisation nach Claudius. Deutsche Zeitschr. f. Chir., Bd. 70, 1903, p. 140.—*v. Mikulicz.* Ueber die jüngsten Bestrebungen, die aseptische Wundbehandlung zu verbessern. Chir.-Kongr. Verhandl., 1898, I, p. 8; im Anschlusse daran: Landerer, Perthes, Döderlein, und Diskussion.—*Minervini.* Zur Katgutfrage. Deutsche Zeitschrift f. Chir., Bd. 53, 1900, p. 1.—*Neuber.* Zur antisept. Wundbehandlung. Chir.-Kongr. Verhandl., 1892, II, p. 76.—*Rosenbach.*

Mikroorganismen bei den Wundinfektionskrankheiten des Menschen, 1884.—*Sarwey.* Bakteriol. Untersuch. über Händedesinfektion. Berlin, 1905.—*Saul.* Ein neuer Versuch zur Sterilisation des Katgut. Arch. für klin. Chir., Bd. 52, 1896, p. 98.—*Schimmelbusch.* Die Durchführung der Aseptik in der v. Bergmannschen Klinik. Arch. für klin. Chirurgie, Bd. 42, 1891, p. 123;—Anleitung zur aseptischen Wundbehandl. Berlin, 1893, 2d edition.—*Sittler.* Die Sterilisation elastischer Katheter. Zentralbl. f. Bakteriologie, Bd. 38, 1905, p. 752.—*Witzel.* Chirurgische Hygiene, Aseptik u. Antiseptik. Die deutsche Klinik, Bd. 8, p 577.

III. GENERAL AND LOCAL ANÆSTHESIA

GENERAL ANÆSTHESIA

Since the discovery of their anæsthetic properties ether and chloroform have contributed immeasurably to the advancement of surgery, and are to-day the most important general anæsthetics.

The anæsthetic properties of ether were discovered first. An American, Crawford Long, performed an operation under ether anæsthesia as early as 1842. Its anæsthetic properties were rediscovered by the chemist Jackson, and were employed by a dentist, Morton, in extracting a tooth; both lived in Boston, the work being done in 1846. Surgeons soon began to use ether (Warren in Boston, Liston in London, Malgaigne in Paris, and Dieffenbach in Berlin, 1847).

Chloroform anæsthesia was discovered by an Edinburgh obstetrician (Simpson). In 1847 he delivered a patient under chloroform after experimenting for some time and comparing the action of chloroform with that of ether. He soon recommended its use, after having employed it in a number of cases.

Like all other anæsthetics, ether and chloroform are poisonous. Their property of rendering patients unconscious and insensitive to pain after the inhalation of certain amounts is a blessing to the patient and a great help to the surgeon. In excess and in susceptible people they may cause death or produce lesions which eventually may end fatally. Therefore they must be used with the greatest caution.

A physician should be intrusted with the administration of either anæsthetic. Attention, practice, and experience, combined with coolness and self-control in emergencies, are required of an anæsthetist, who should meet rapidly and effectively any emergency which may arise.

CHAPTER I

CHLOROFORM ANÆSTHESIA

Physical Properties of and Tests for Chloroform.—Chloroform, $CHCl_3$, formyltrichloride, trichlormethane, is a clear, colorless, volatile fluid with

a peculiar aromatic odor and sweet burning taste. It boils at 142° F. It is decomposed by daylight and air into hydrochloric acid, chlorine, free formic acid, phosgen, etc., and should therefore be kept in dark colored, tightly closed bottles. By the addition of one per cent absolute alcohol the decomposition of chloroform may be prevented.

Unpleasant symptoms have followed the use of impure chloroform; death has been produced by its decomposition and substitution products. Only reliable, pure preparations should therefore be used for anæsthesia. [In America Squibb's chloroform, especially prepared for anæsthesia, is generally preferred. It comes in small stained glass bottles, which may be conveniently used for dropping the chloroform. Although a number of manufacturers have placed their chloroform upon the market, Squibb's is still generally employed.]

Hepp's smelling test may be employed to determine whether chloroform is pure or not. It is used in the following way: Some chloroform is dropped upon white filter paper and allowed to evaporate. If the chloroform is pure the paper has no odor; if impure, a penetrating, rancid odor remains, which is produced by the decomposition products.

The Action of Chloroform Vapor when Inhaled.—Inhaled chloroform vapors pass into the blood through the alveoli of the lung, and are then carried to all the organs, including the central nervous system. The paralysis of the nerve centers begins in the great lobes of the brain, then attacks the cerebellum, and finally the spinal cord, sensation being lost before motion. The centers in the medulla retain their function the longest; if these are paralyzed death occurs.

Chloroform in large amounts or with susceptible people paralyzes the ganglia situated within the heart which control the heart beat, and is toxic for heart muscle itself. A fall of blood pressure follows the paralysis of the vaso-motor center, and the heart has to perform an excessive amount of work and becomes exhausted (Kappeler). The direct action of chloroform upon the nasal and laryngeal mucous membrane may cause a reflex respiratory paralysis and influence the heart beat by irritation of the trigeminal branches supplying the nasal mucous membrane, and of the superior laryngeal nerve, supplying the laryngeal mucous membrane, and of the vagus.

Like any other anæsthetic, chloroform passes into the blood of the fœtus and into the milk of the nursing mother.

Chloroform is excreted by the lungs, the skin, and the kidneys.

It produces a transitory fatty degeneration of the myocardium, liver, and kidneys. These fatty changes may become more extensive and finally cause death, especially if the organs were previously diseased.

Preparation of Patient for General Anæsthesia.—Every patient should receive special preparation for an anæsthetic.

Washing the mouth and cleaning the teeth reduce the dangers of aspiration pneumonia.

If possible the patient's stomach should be empty. This prevents the accidents resulting from vomiting, and lessens the distress and vomiting following the anæsthesia. Patients should not be allowed to take food for six hours before the operation, and if in an emergency an anæsthetic must be given shortly after meal time, the stomach contents should be removed by a stomach tube.

Tight clothing, corsets, abdominal binders, and collars should be removed to prevent interference with respiration. The shirt should be left open at the neck, and foreign bodies (artificial teeth, tobacco, and candies) should be removed from the mouth.

[Magaw, in a review of over fourteen thousand anæsthesias induced in the Mayo clinic at Rochester, Minn., gives some very practical suggestions regarding the administration of an anæsthetic. She employs almost exclusively the "open or drop method," and prefers ether to chloroform. She describes the method as follows:

"Patients usually walk into the operating room and mount the table with assistance. All foreign bodies, such as artificial teeth, chewing gum, etc., are removed. The hands are fastened loosely across the chest with a wide gauze bandage, to prevent the arms falling over the sharp edges of the table, an accident which often causes musculospiral paralysis. A pad of moistened cotton is placed over the eyes to protect them from the anæsthetic. If, during the course of the administration, some of the anæsthetic should fall in the eye, drop a few drops of castor oil into the conjunctival sac to prevent the conjunctivitis that would otherwise follow.

"It is a mistake to think that the same elevation of the head will do for all patients. The anæsthetist should elevate the chin to such a position as not to bend the neck too far back or approximate the chin too near the sternum. Proper elevation of the head will relax all tissues of the neck and give more freedom in breathing. This also can be said of the jaw. Holding the jaw forward and keeping it in position, so that the patient gets the greatest amount of air possible, is an important feature in giving an anæsthetic. While too much stress cannot be laid on this necessary requisite in giving an anæsthetic, all jaws cannot be handled in the same manner. When a patient has removed a double set of false teeth, the tongue will often cleave to the roof of the mouth during the administration, and raising the jaws sets the gums so firmly together that most of the air is shut out, and this may not be noticed until the patient is cyanotic. We have found in this class of cases that if the jaw is held but slightly up and forward, and the thumb at the same time inserted between the gums, thereby holding the tongue down,

faulty respiration will be corrected at once and color restored. This is one of the instances where holding up of the jaw too firmly may be overdone.

" All patients have been anæsthetized on the operating table in the operating room, and the preparation of the patient was going on at the same time. Experience has taught us that preparation of the patient while going under the anæsthetic is one of the important factors in producing a rapid surgical narcosis, for it diverts his attention and much less anæsthetic is required. It matters not in what position the patient must be for operation, we fix him accordingly, and the preparation is begun at the same time as the anæsthetic, and we feel certain that this procedure enables us to hasten narcosis.

" In the Trendelenburg position, where the preparation is in progress during the administration of the anæsthetic, the deep respiration, etc., empties the pelvis, so that by the time the operation is started the small bowel will be found in the upper abdomen and out of the way, and may be packed off. We have found this practice more helpful to the surgeon than placing the patient in position after the completion of narcosis.

" In giving an anæsthetic for this class of surgery, the skill and patience of the anæsthetist is tried to the extreme, and the patient must be anæsthetized, but not too profoundly. Patients having an acute peritonitis, as is so often found in this class of cases, require a much larger amount of anæsthetic to produce relaxation of abdominal muscles. When the patient is prepared during the administration of an anæsthetic, there is no time lost, the surgeon and his assistant being ready by the time the patient is surgically anæsthetized. Another important reason for anæsthetizing the patient on the operating table is that in lifting and shifting a patient about he is apt to regain consciousness, with vomiting, etc., and the anæsthetist cannot be positive of the condition of his patient. Should ether produce difficult breathing, profuse secretion of mucus or cough, lift the mask from the face, allow a liberal amount of air, and then continue with ether. In giving plenty of air when needed and less anæsthetic, we have found little use for an oxygen tank, a loaded hypodermic syringe, or tongue forceps. It is far better for the anæsthetist to become skillful in watching for symptoms and preventing them than to become proficient in the use of the three articles above mentioned. An acute cold is a contraindication to any anæsthetic, but as soon as a cold becomes chronic there is not much danger from etherization, and instead of operating during an acute cold and giving chloroform (unless in an emergency), we wait a few days until the acute attack has passed, and then they are as good subjects for ether as for any other anæsthetic. Chronic bronchitis is often improved by an anæsthetic."]

The anæsthetist should gain and retain the confidence of the patient, who should not be permitted to see the instruments or any blood-stained sheets or dressings. Patients frequently become excited and frightened upon entering the operating room, and for this reason it is the custom in a number of clinics to begin the anæsthesia in a small room immediately adjacent to it.

Position in which Patient should be Placed for General Anæsthesia.—Before the administration of the anæsthetic is begun the patient should be placed in a comfortable horizontal position; the head, which should neither be extended nor markedly flexed, lying on a small roll or pillow. The head and trunk of weak, anæmic patients should never be elevated, especially if a major operation is to be performed. In these cases the head and trunk should be lowered, as in this way the dangerous effects of cerebral anæmia may be avoided. If the position is comfortable, the patient should be asked to close his eyes, to breathe naturally, and to avoid swallowing saliva. In order to distract the attention of the patient he may be asked to count.

The Different Stages of General Anæsthesia.—Chloroform anæsthesia, like all general anæsthesias, passes through four stages:

1. Initial stage.
2. Stage of excitement.
3. Stage of deep anæsthesia.
4. Stage of awakening.

1. After the first few inspirations most patients begin to hold their breath and swallow air. The sweet taste and odor of chloroform vapor is disgusting to many, especially to children and patients who have been anæsthetized a number of times. During this stage patients frequently experience a sensation of suffocation and, crying for air, tear the mask from the face. Soon spots appear before the eyes, the patient becomes dizzy and has unpleasant, often terrifying dreams, the ears ring and the heart pounds. The face becomes reddened, the patient talks incoherently, alternately laughs and cries and acts like a drunken man. Consciousness and sensation are gradually reduced, and then rapidly lost. The salivary secretion is increased, the pulse is rapid and full, the respiration rapid but deep; the pupils are dilated but react to light, and when the eyelids are raised and the cornea touched a wink may be elicited. The reflex irritability is still considerably increased, and a painful examination or sometimes too early sterilization of the field of operation may excite violent and often dangerous struggles.

2. The stage of excitement is the more pronounced the more unevenly and rapidly the anæsthetic is given. Children do not pass through this stage; women frequently do not. It is rarely absent in men, being most marked in the strong and vigorous and in alcoholics. It begins with contraction of the muscles of the trunk which lasts but a short time, sud-

den extension of the head and extremities, and it may end quickly. If the stage is more pronounced the patient acts as though insane, clutches the anæsthetizer, flings his arms about, distorts the face, tries to get up or throw himself from the table. Urine, gas, and fæces may be discharged by the contraction of the abdominal muscles. The patient sings, shrieks, and moans, expectorates into the mask, breathes deeply and rapidly or may hold his breath, and then his face becomes cyanotic. The jaws are closed by a spasm of the masseters, the eyes are rolled about, the pupils are slightly dilated, and react but little to light.

3. After a few minutes the contracted muscles become relaxed, the raised hand falls, the face becomes smooth, and the stage of deep anæsthesia in which major operations may be performed is reached. As a rule it requires about ten minutes to bring the patient to this stage of anæsthesia. Finally the masseters relax, the jaw and the tongue drop backward, and the patient snores; the face becomes pale, the pupils contract and react but little to light. The reflexes are abolished, and if the cornea is touched there is no reaction. The pulse becomes slow, the respirations regular and superficial.

Chloroform does not interfere with uterine contractions, but stops the contraction of voluntary muscles which supplement these. It is extensively employed by obstetricians.

4. Some patients recover from an anæsthetic more rapidly than others, and they behave differently. Rolling of the eyes is the first sign of awakening. As a rule, children after crying or vomiting fall asleep again. Adults vomit, then become excited and laugh or cry (particularly alcoholics and hysterical patients); some experience a sensation of well-being, following the excitement of the operation. Most men desire to sleep and be left alone; they are irritable and complain of headache and discomfort, which is increased by pain in the wound.

The operator should pay as much attention to the length of time the anæsthetic is given as he does to operative technic, asepsis, and the control of hæmorrhage. On an average, anæsthesia should not be continued for more than an hour; even in the most difficult cases not for more than two hours. The shorter the anæsthesia, the greater the chances of recovery from major operations.

It is difficult to measure the amount of anæsthetic given when an ordinary mask is used. During expiration, especially in the stage of excitement, the chloroform evaporates. The amount can be measured only when an apparatus especially constructed for the purpose is used.

The administration of chloroform is an art. The anæsthetist should devote his entire attention to the administration of the anæsthetic, and should pay no attention whatever to the operation. If the operation is upon the head or neck, he should sterilize his hands, put on a sterile

gown, use a sterile mask, and wrap the chloroform bottle in sterile gauze. He should note the appearance and color of the face, test the pupillary or corneal reflexes, watch the breathing, note the respiratory movements

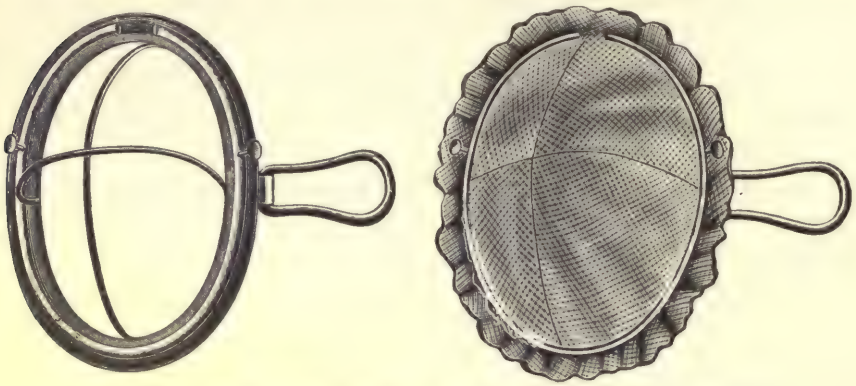


Fig. 51.—The Chloroform Mask of Schimmelbusch. (From Dumont.)

of the abdominal wall and take the pulse, if this is not delegated to a colleague. Rather intrust the anæsthetic to an experienced nurse than to an inexperienced physician. Another physician besides the operator should be present to assist in case of accident, and, as a precaution, to act as a witness if accusations of assault or theft should be made.

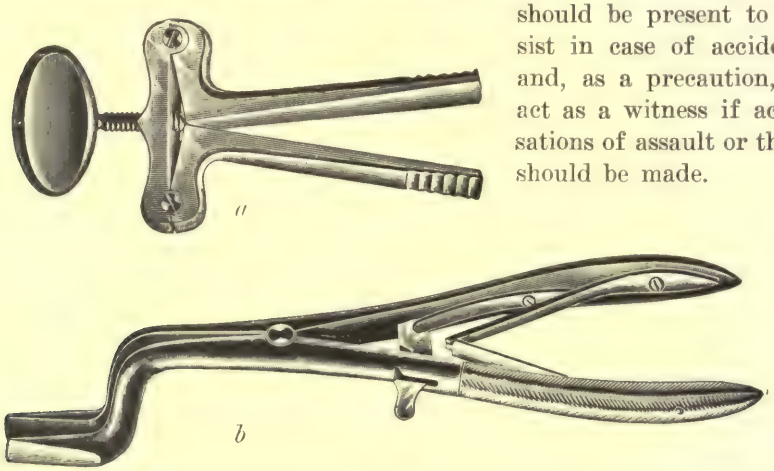


Fig. 52.—*a*, Heister's Mouth Gag (from Dumont); *b*, The Koenig-Roser Mouth Gag (from Dumont).

Everything that may be required should be near at hand. The Schimmelbusch mask is usually used when chloroform is administered. It consists of a wire frame which may be sterilized, over which gauze is fastened. Chloroform is kept in small brown bottles. Different provisions are made for dropping it. The Heister, Koenig-Roser, or the

newer P. von Bruns mouth gag may be used. Tongue forceps, steel sponges, a towel, and a basin to catch the vomitus should be provided.

A clean handkerchief may be used instead of a chloroform mask. It should not be laid directly upon the face, for the chloroform may burn the skin, even if vaseline has previously been applied. A stand-up collar, buttoned in front and bent into an oval, may be placed upon the face and a handkerchief spread out over it.

Apparatus for Determining Amount of Chloroform Administered, etc.—The different apparatus used in large clinics for the administration of chloroform have some advantages, as the amount of chloroform can be accurately measured and the amount of oxygen given with it controlled, and thus if the breathing is deep, the danger of giving too much chloroform is reduced. The Junker apparatus, improved by Kappeler (Fig. 53), is provided with a rubber bag, and by pressing it a mixture of chloroform and air is supplied to the patient. The Wohlgemuth and Roth-Dräger apparatus are too large for practical purposes and are expensive. They supply a mixture of chloroform and oxygen, and in case of asphyxia oxygen alone may be given. Any apparatus has the disadvantage that the anæsthesia is induced slowly; often it does not pass beyond the stage of excitement and must be continued with the ordinary mask.

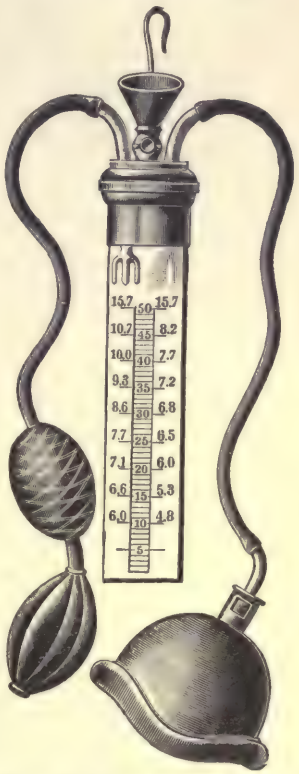


FIG. 53.—JUNKER'S APPARATUS AS MODIFIED BY KAPPELER. (From Dumont.)

Dropping of Chloroform.—In administering chloroform a dry mask should be laid upon the face, covering the mouth and nose, and then chloroform should be slowly dropped upon it, from 10 to 60 drops being given in a minute, depending upon the age of the patient. This may be increased up to even twice the amount, until the stage of excitement begins. A saturated mask should never be used, because of the danger of inducing a violent stage of excitement and causing reflex cardiac or respiratory paralysis. This is often done, even by experienced anæsthetists. If the patient is greatly excited the mask should be removed for one, two or more minutes to avoid administering too much when he begins to breathe deeply. After such interruptions the anæsthesia should be continued. About 60 drops

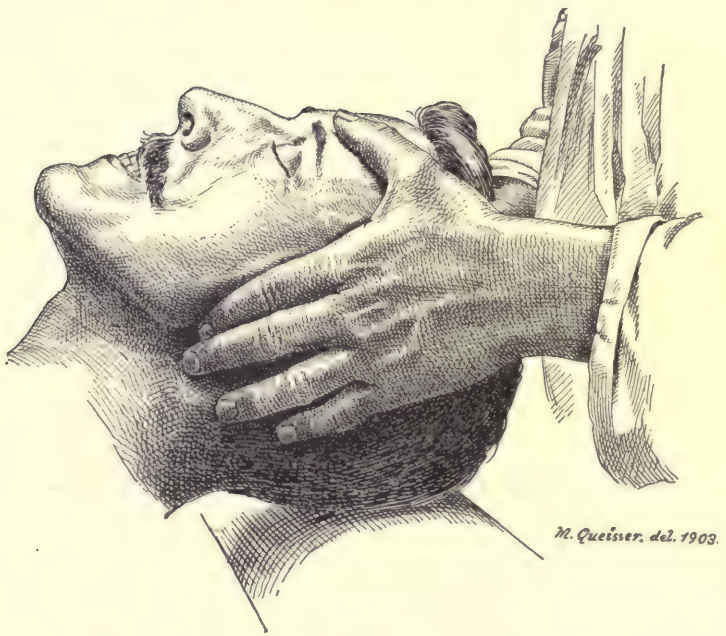
should be given in a minute until the patient is relaxed. An experienced anæsthetist regulates the dropping of the chloroform by intuition or the carotid pulse. If the pulse is rapid one drop is given for every two to three beats; if slow, one drop for each beat. The chloroform bottle should be held near the mask, so that if the patient moves the chloroform will not be dropped upon the skin or in the eye. The skin has been burned, and corneal opacities have been caused in this way. Such results may be avoided if the chloroform is wiped off the skin and the eye irrigated immediately after such an accident. During the stage of excitement the patient should not be forcibly restrained, as this merely increases the excitement, and fractures and dislocations may be produced.

Different Methods of Holding the Jaw Forward.—If in deep anæsthesia the breathing becomes stertorous and labored the jaw should be drawn forward, and in adults, as a rule, it should be held in this position, for as the jaw drops backward, carrying the tongue with it, the relaxed epiglottis is pushed downward, closing the opening of the larynx. In pushing the jaw forward the von Esmarch-Heiberg method may be employed: The flat hand is placed over the ear, the tips of the index fingers behind the angle of the jaw and the thumbs upon the temporal or frontal regions, and the jaw is then gently pushed forward by the index fingers until the lower teeth project beyond the upper. The different steps in this method, the correct and false, are represented in Figure 54, *a* and *b*. Pressure should not be made upon the internal jugular vein, and too much force should not be used, as the jaw may be dislocated or a contusion about the angle of the jaw produced. If the patient's head is turned to the right, the jaw may be held in this way by the left hand alone, and the right hand be used for giving the anæsthetic. If this does not suffice to overcome the embarrassment of respiration, the mouth should be opened with the mouth gag and the tongue drawn forward. If it is necessary to hold the tongue forward for some time, it is advisable to pass a heavy silk ligature through it; in this way the injury produced by tongue forceps may be avoided.

If the anæsthetist stands in front of the patient the jaw may be drawn forward by the Kappeler method: The thumbs being placed upon the maxillæ beside the nose, and the flexed index fingers behind the angle of the mandible, and the jaw drawn forward.

Stage of Anæsthesia Which should be Maintained During an Operation.—It is the duty of the anæsthetist to keep the patient in the quiet stage of anæsthesia, permitting him neither to awake nor to be overcome by a fatal paralysis of the respiration or of the heart. The character of the pulse and respiration, the color of the face, and the condition of the pupil indicate the condition of the patient.

a



b

FIG. 54.—PUSHING THE LOWER JAW FORWARD. *a*, Incorrect method; the jaw is not pushed far enough forward, the internal jugular vein is compressed; *b*, the correct method.

To test the pupillary reflex both upper lids should be raised by the second and third fingers, after they have been tightly closed for a second. In the first and second stages of anæsthesia the pupil is dilated and reacts slowly, because of irritation of the cervical sympathetic. In the beginning of the third stage the pupil becomes contracted but reacts quickly. As the anæsthesia becomes deeper the pupil becomes narrower and reacts less rapidly, until finally, as the result of irritation of the oculomotor nerve, it becomes fixed and pinhead in size. When the pupil is in this condition the stage of deepest permissible anæsthesia has been reached. Anæsthesia should not be carried to this so-called normal point, but should be maintained in that stage in which the pupils are contracted, but react to light. If the anæsthesia is carried beyond the normal point, the pupils will dilate, because of paralysis of the sphincter iridis, and will not react, and cardiac and respiratory paralysis will quickly occur. It is impossible to give any definite rules by which anæsthesia may be maintained at the proper stage. Each patient behaves differently; some require a long, some a short time, to reach this point. (The danger zone varies, Czempin.) It is so rapidly reached and passed by many that it may be overlooked by the anæsthetist. In children and weak adults two to four drops of chloroform in a minute are enough to maintain deep anæsthesia. In adults about twenty drops are required, but sixty to eighty drops may be required, especially for men.

Interrupted Anæsthesia.—It is most difficult to give an interrupted anæsthesia, as is often required in operations about the head and face, and to avoid the dangers described above. Awakening with vomiting and the dangerous stage of inactive dilated pupils quickly follow each other, if the anæsthetist is not skilled and attentive. In these cases the patient should be slowly anæsthetized until the pupils contract, and then the mask should be removed so as to expose the field of operation. As soon as the reflexes return and the pupils dilate again, more anæsthetic should be given.

The disadvantages of this interrupted anæsthesia may be avoided if the canula devised by Salzer is employed. This bent canula (Fig. 55)

FIG. 55.—SALZER'S CHLOROFORM CANULA TO BE INSERTED INTO THE MOUTH IN OPERATIONS UPON THE FACE AND MOUTH. (From the Clinic of von Mikulicz.)

is inserted into the mouth cavity at the angle, and is then attached to the Junker-Kappeler apparatus. [A rubber catheter or piece of rubber tubing may be passed through the nose into the nasopharynx, and the anæsthetic administered through it after being attached to the apparatus

above mentioned. It is much simpler and more convenient than the special canulæ which have been devised.]

Pupillary and Corneal Reflexes.—The testing of pupillary reflexes is unreliable in hysterical patients, as frequently the pupils do not contract. The pupillary reflex is also unreliable in all conditions characterized by myosis, such as morphinism, nicotine poisoning, locomotor ataxia, paretic dementia, diseases of the corpora quadrigemina, meningitis. Morphine is frequently given before a general anæsthetic is administered, and in these cases it should be remembered that no significance whatever can be attached to the pupillary reflex. [It is the belief of the editor that too much significance has been attached to both the pupillary and corneal reflex. The latter is unreliable and results following its elicitation are often distressing; as severe conjunctivitis may follow the injury inflicted on the corneal epithelium. It is much more essential that the anæsthetist note carefully the respiration and the color of the patient. Cyanosis means danger, and the anæsthetic should be withdrawn when the patient becomes dusky. Any interference with respiration should be instantly recognized and relieved.]

Fig. 56.—Chloroform Apparatus for Administering an Anæsthetic Through a Tracheotomy Tube. (From Dumont.)

It is occasionally necessary to administer an anæsthetic through a Hahn or Trendelenburg tampon canula. The apparatus represented in Figure 56, which consists of a metal funnel and a tube, may be used for this purpose. The funnel is covered or filled with gauze, upon which the anæsthetic is dropped and the tube is attached to the canula.

The character of the pulse should be carefully noted during the entire anæsthesia, as it indicates dangerous overexertion of the heart and is a good index as to the general condition of the patient, the effect of the anæsthetic, and the operation.

After Effects of Chloroform Anæsthesia.—The after effects of chloroform anæsthesia vary in different patients, being largely dependent upon the amount of chloroform used. They occur most frequently after long or frequently repeated anæsthesias. Nausea and vomiting are the most constant, as the gastric mucous membrane is irritated by the saliva swallowed at the beginning of the anæsthesia and by the chloroform vapor. The so-called "chloroform distress" persists, as a rule, for twenty-four hours, but may last from two to three days and render the patient very weak. Icterus frequently follows chloroform anæsthesia as the result of changes in the liver, and the destruction of red blood corpus-

cles. The stomach symptoms may sometimes be prevented if a towel moistened with vinegar is applied to the nose immediately after the patient awakes (Lewin). Post-anæsthetic nausea and vomiting may be prevented by washing out the stomach with lukewarm water or a one or two per cent soda solution, and by withholding food. If not nauseated the patient may be given some nourishment in four or five hours after awakening. It is best to begin with teaspoonfuls of tea, warm soup, or red wine. Coffee and mineral waters should be avoided, as the former causes heartburn, the latter incite vomiting. If the vomiting continues for some time, rectal feeding is indicated (milk with eggs; in case of collapse, warm red wine with cloves). The swallowing of air frequently incites vomiting. A small dose of morphine frequently controls the vomiting. The administration of favorite articles of food should be discouraged.

Patients, as a rule, do not sleep the night following the operation. Nervous irritability, pain in the wound, unusual positions in bed, nausea, etc., are the usual causes. Weak patients should be given morphine to induce sleep. Hysterical and melancholic patients may have attacks of mania; the nervous symptoms may last several days.

Post-Anæsthetic Palsies.—The so-called post-anæsthetic palsies are mostly peripheral. The musculospiral nerve may be paralyzed if the arm is allowed to hang over the sharp edge of the operating table. The nerves of the brachial plexus may be pressed upon and contused by the clavicle or the head of the humerus if the arm is strongly abducted. These palsies may last for some time. Central paralysis, the result of a hæmorrhage into the brain during the stage of excitement, is rare.

If the patient does not recover from the anæsthetic, he becomes progressively restless, the pulse becomes weak and fluttering, the vomiting becomes uncontrollable, the urine contains albumin, the urinary secretion diminishes and uræmic symptoms develop, and death from collapse may follow in from one to three days.

If the post-mortem examination discloses fatty degeneration of heart muscle, fatty degeneration and necrosis of the cells of the kidney and liver, death should be attributed to the late effects of chloroform. It is often difficult, however, to exclude in these cases other causes of death, such as operative shock after long operations, anæmia following severe hæmorrhage, and acute general infections.

Broncho-pneumonia and bronchitis are rarely caused by chloroform, and when they occur should be regarded as produced by the aspiration of saliva or vomitus, if pulmonary embolism can be excluded. Broncho-pneumonia and bronchitis follow quite frequently abdominal operations in which pain prevents coughing. They also follow anæsthesias administered in rooms where there are unprotected kerosene or gas flames, as

the chloroform is decomposed into phosgen gas, hydrochloric acid, chlorine, and other bodies which irritate the lungs. A peculiar odor, irritation of the throat of the patient and that of the surgeon, and a haziness about the operating table indicate that this decomposition is occurring. If this happens, the windows and doors should be opened immediately.

CHAPTER II

ETHER ANÆSTHESIA

Physical Properties of Ether.—Ether, sulphuric ether, $C_4H_{10}O$, is a clear, diffusible, colorless, inflammable fluid with a peculiar odor; is very volatile, boils at 95° F., and has at 59° F. a specific gravity varying from 0.720 to 0.722.

Ether should be kept in tightly sealed cans or brown bottles and protected from the light and air in order to prevent decomposition. Only pure preparations of ether should be used for anæsthesia. Impure ether, after evaporation in a watch crystal, leaves a residue which colors blue litmus paper red.

Differences between Chloroform and Ether Anæsthesia.—Ether vapor when inhaled has much the same effect as that of chloroform; the four following differences, however, should be noted: 1. Ether has less effect upon the heart than chloroform, and there is less danger of death from cardiac paralysis when ether is employed. 2. Ether, as a rule, raises blood pressure, while chloroform lowers it. 3. Ether has a wider danger zone than chloroform—that is, the difference between the anæsthetic and fatal dose of ether is greater, and therefore there is less danger of sudden collapse during ether anæsthesia. 4. Ether irritates the mucous membranes of the respiratory passages, especially those of the mouth, nose, and pharynx. Lesions of the lung frequently follow its use. The changes in the lung may be due to the aspiration of saliva, the secretion of which is increased, as well as to the direct, irritating action of the ether vapor. This is one of the disadvantages of ether as compared with chloroform.

Ether is inflammable, therefore a thermocautery cannot be employed about the mouth and face when it is used, and it cannot be given when an unprotected flame is near by. If the lamp hangs high above the operating table it may be used, as its fumes sink. It is not decomposed as chloroform is by a naked flame.

Preparation of Patient for Ether Anæsthesia.—A patient should receive the same preparation for ether anæsthesia as has been described for

chloroform. Ether anæsthesia passes through the same stages as chloroform; the behavior of the reflexes, especially the pupillary reflex, and the after effects are very similar. The pulse remains full, and is even increased in volume and rapidity, therefore the color of the face remains normal, and vessels cut during an operation bleed profusely. An increased amount of saliva is formed, which interferes with operations about the mouth and adjacent to it and embarrasses respiration, giving a gurgling character to the breathing and causing cyanosis. Death on the table from suffocation is, however, less likely to occur than subsequent pneumonia due to the aspiration of saliva and its irritating action upon the respiratory mucous membrane.

An anæsthetist who has a tendency to catarrh and is incited to severe spells of coughing by the inhalation of small amounts of ether, is also exposed to the dangers of pneumonia. The administration of ether demands the same careful attention that should be given to that of chloroform.

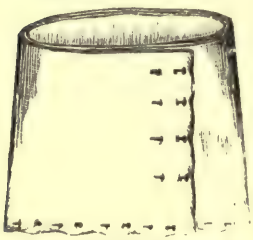


Fig. 57.—Cloth and Paper Cone.

Methods of Administering Ether: Open and Closed.—[Ether may be administered by either the open or closed method. The former is very extensively employed in America at present, and Magaw, who had such a large experience with ether, prefers it to any other. In the open method the ether is dropped from a four or eight ounce ether can, fitted with an ordinary cork with a groove on either side.

One groove should be filled with cotton or absorbent gauze, which should extend out of the can about one inch. The size of the drop can be regulated by cutting the gauze or cotton in a certain way and

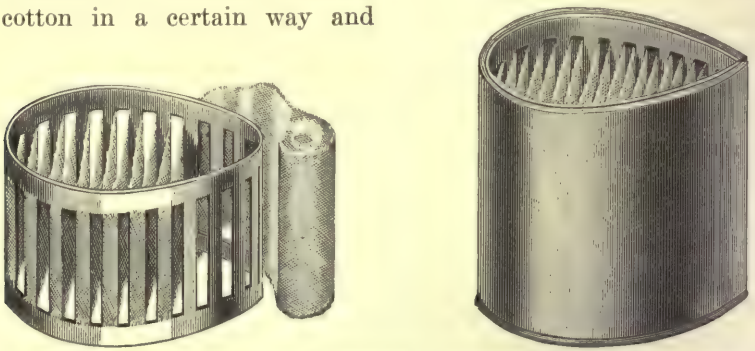


Fig. 58.—Allis's Inhaler.

regulating the cork. Magaw usually fixes two cans, one with a large dropper, and uses it until the patient is fully under the anæsthetic, and

then changes to the other can with the small dropper, and continues its use during the operation.

An improved Esmarch inhaler, covered with several layers of gauze or two thicknesses of stockinet, is employed. Of course the gauze or stockinet should be changed after each anæsthesia, and the frame should be boiled. The ether should be dropped as slowly and as carefully as if it were chloroform until the patient's face is flushed. Then a few layers of gauze are added, and the ether is given in larger drops and more rapidly until the patient is surgically anæsthetized, at which time the gauze may be removed.]

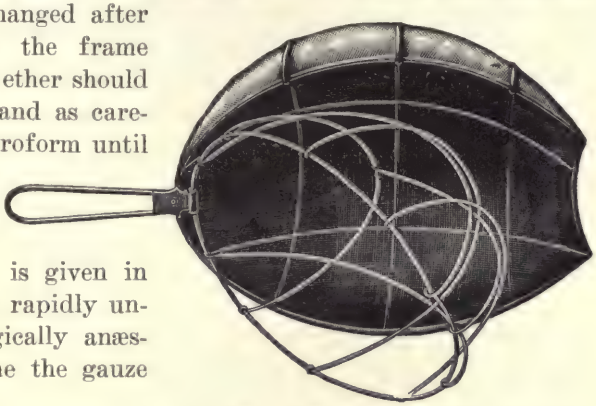


FIG. 59.—ETHER MASK AFTER JULLIARD-DUMONT.

In the closed method a cardboard mask covered with muslin or the Julliard mask, improved by Dumont, is used. If the cardboard mask is used, several layers of gauze are inserted upon which the ether is dropped. The Julliard mask consists of two metal frames placed one over the other and connected by a hinge, the outer frame being covered by oilcloth. Several layers of sterile gauze are placed between the two frames, and a flannel pad upon which the ether is poured is attached to the inner frame (Fig. 59). In beginning the anæsthesia about 20 g. of ether are poured upon the flannel, and the mask is then gradually placed upon the face. After one or two minutes the mask is removed, the same amount of ether is poured on the flannel, and it is reapplied again. The mask is then surrounded by a towel to prevent the escape of ether (Fig. 62).

FIG. 60.—GRADUAL APPLICATION OF MASK TO FACE. (From Dumont.)

The towel is removed when the stage of surgical anæsthesia is reached. The mask is removed from time to time, depending upon the condition of the patient, and ether is poured upon it if necessary. The air has free access to this mask, there is no accumulation of carbon dioxide and no reduction of oxygen, therefore the blood is well aërated (Dumont).

If a saturated mask is held tightly over the patient's mouth and nose at the beginning, he experiences a sense of suffocation, coughs, becomes restless, and, after a few inhalations, suffocation and reflex cardiac or respiratory paralysis may ensue. For this reason this method, which has been rightly called the suffocating method, should never be used.

Administration of Morphin and Atropin before Ether Anæsthesia.—The mask should be momentarily withdrawn during the stage of excitement when the patient holds his breath or the inspirations become very deep. It is much more difficult to overcome the stage of excitement with ether than with chloroform. It is recommended for this reason that adults be given before ether anæsthesia $\frac{1}{8}$ gr. morphin, or, according to Dastre, $\frac{1}{8}$ gr. morphin and $\frac{1}{100}$ gr. of atropin, as the latter diminishes secretion.

Fig. 61.—Lifting the Mask to Inspect Face and Permit of Free Access of Air. (From Dumont.)

During the anæsthesia the mask should be removed frequently, in order that the color of the face may be noted, the pupillary reflexes tested, and the saliva wiped away. If the pupil reacts rapidly, more ether should be poured upon the mask, which should then be replaced.

Inhalers by Which Amount of Ether may be Regulated.—A number of different inhalers have been introduced by which the amount of ether administered can be controlled. The Clover, Ormsby, Squibb, and Bennett inhalers are all good, and are eminently satisfactory when used by one accustomed to them. If, however, one has an intimate knowledge of anæsthesia, he can give ether just as satisfactorily by the open or closed method as he can with the inhalers above mentioned. They have

not been generally adopted, although expert anæsthetists who have become accustomed to either one of the inhalers above mentioned speak of them very highly.

Von Arnd has devised an apparatus with a compressible bag for operations about the face and mouth. A curved metal end piece attached to a piece of rubber tubing which is connected with the bag is introduced into the mouth. A mixture of air and ether is blown into the mouth by compressing the bag. As the ether is still further diluted by the inspired air there is but little irritation of the mucous membranes.

FIG. 62.—SURROUNDING THE MASK WITH A TOWEL. (From Dumont.)

Increased Secretion of Saliva and Mucus during Ether Anæsthesia.—During ether anæsthesia the respiratory passages must be kept free, as in this way the dangers of suffocation and aspiration pneumonia may be avoided. Gurgling respiration and cyanosis indicate danger, which is increased if the saliva flows into the larynx. The flowing of saliva into the pharynx and larynx may be prevented if the head is held on the side. The mouth should be kept open, and if there is an excessive amount of mucus and saliva the mouth should be cleaned out with gauze.

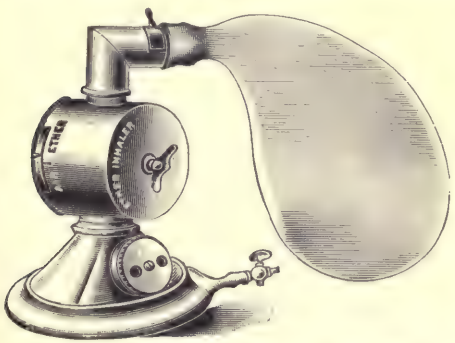


FIG. 63.—BENNETT'S ETHER INHALER.

The pharynx, if necessary the larynx, may be wiped out with sponges on forceps. The latter should not be employed unless absolutely necessary, as the mechanical irritation

produced by them often leads to the secretion of more mucus. If the jaw drops backward and interferes with respiration, it should be pushed and held forward. The tongue should be drawn forward if it drops backward and closes the larynx.

Incomplete Ether Anæsthesia for Short Operations.—Incomplete ether anæsthesia may be used for short operations. Kronecher recommends that in these cases the anæsthesia be stopped immediately after the stage of excitement, while Sudeck places the mask directly upon the face and performs minor operations after the first few inspirations in the so-called "ether drunk." The after effects of ether are about the same as those of chloroform, and should be treated in the same way.

Lung Complications following Ether Anæsthesia.—Broncho-pneumonia, bronchitis, and œdema of the lungs are more common after ether than after chloroform anæsthesia. The lung complications are due to the aspiration of mucus and saliva, the use of impure ether, and too great concentration of the ether vapor. Other factors, which are also present in chloroform and local anæsthesias, such as cooling of the surface of the body, the inability to cough and expectorate after abdominal operations, are also contributing factors.

The symptoms of lung complications usually develop on the second or third day. Frequently they run a mild clinical course, yet they may end fatally, especially if the lungs were previously diseased (bronchitis, emphysema, tuberculosis) or if they develop in old and feeble patients.

Ether and chloroform have about the same effect upon a preëxisting nephritis, the albuminuria following ether anæsthesia when the kidneys were previously sound is more rapidly recovered from.

Central Anæsthetic Palsies.—Central anæsthetic paralyses are more to be feared when ether is used, as it raises blood pressure. They occur only in patients who at the time the ether was administered had a high blood pressure; most commonly in patients suffering with arteriosclerosis, interstitial nephritis, and lead intoxication.

Venous Thrombosis.—Thrombosis of the large veins of the pelvis and lower extremities is another complication which may follow the use of ether. It is often associated with inflammatory changes in the pelvis, other local and general causes, such as toxæmia, anæmia, cardiac weakness, etc.

Ether increases the coagulability of the blood, and at times when injected, even in small amounts, into the veins of animals, produces extensive thrombosis (Hanau, Ribbert). According to the experimental researches of Lexer and Mulzer, thrombi are found in the small blood vessels and capillaries, especially of the lungs and kidneys, after the inhalation of either chloroform or ether. The thrombosis becomes more extensive the longer the anæsthetic is administered.

CHAPTER III

NITROUS OXID ANÆSTHESIA

[NITROUS OXID, N_2O, is usually obtained by heating ammonium nitrate, which decomposes at an elevated temperature and forms water and nitrous monoxid ($NH_4NO_3 = 2H_2O + N_2O$). The product is washed by passing through water, which soon becomes saturated with the gas. The gas is kept in retorts or tanks obtained from manufacturers, in which it is reduced to a liquid form by strong pressure. The bag from which the gas is administered is filled, and the amount of gas regulated by a stopcock.

Anæsthesia is rapidly induced by nitrous oxid, and the gas must be given continuously or intermittently, the mask being removed for short intervals, when the patient becomes blue, in order to maintain surgical anæsthesia.

Nitrous oxid gas, the safest of the anæsthetics, has been left until recently to the dentist and for minor operations, although years ago the feasibility of employing it for prolonged anæsthesia was thoroughly demonstrated by Bert, Andrews, and others.

In the last eight or ten years nitrous oxid has been extensively used by the general surgeon in the sequence of nitrous oxid and ether. In this I believe it has no very great value, except that it offers an agreeable anæsthesia to the patient.

During the last three or four years I have been employing nitrous oxid and air as a general anæsthetic in an increasing number of patients, and I have been so much impressed with its value and possibilities of wide application that I feel warranted in urging its more general use.

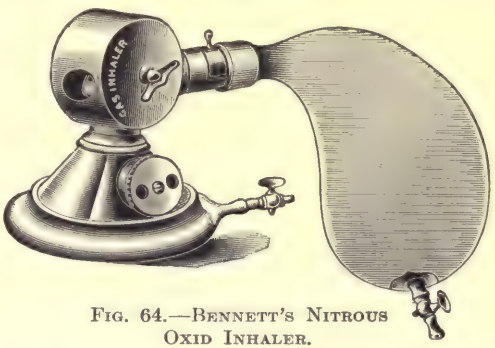


FIG. 64.—BENNETT'S NITROUS OXID INHALER.

Roughly speaking, chloroform anæsthesia has a mortality of 1 in 2,000; ether, 1 in 5,000; and nitrous oxid gas, 1 in 50,000 to 1 in 100,000. Gas is the most agreeable anæsthetic to take, and is the most rapid anæsthetic, taking usually about sixty seconds. It is seldom followed by nausea or vomiting, pneumonia or bronchitis or nephritis or secondary changes in the tissues. If properly administered with air, an anæsthesia of a half hour or an hour can be secured.

I began using it in cases where ether and chloroform were specially contraindicated, as in operations on the kidneys, such as nephrotomy for anuria, abscess, etc. I then extended it to kidney stone operations and nephrectomies. And finding how easy it was to maintain satisfactory anæsthesia for long periods, I have gradually increased the range of its use until now I am employing gas in a large proportion of my general cases.

It is the anæsthetic of choice in reducing fractures and dislocations, in opening abscesses and felons, in breaking up adhesions in joints, in draining empyemas and lung abscess, in exploratory laparotomies, in gall bladder work, removing stones and drainage in kidney work, nephrotomy, nephrectomy, and nephrolithotomy, in bladder work, suprapubic cystotomy for stone and in suprapubic prostatectomy, in draining appendiceal abscesses and cases of general peritonitis, in colostomy, in gastrostomy, in enterostomy, in repair of typhoid perforation, in repair of perforating gastric and duodenal ulcers, in hernia operations, especially for relief of strangulated hernia, in varicocele, in open operation for hydrocele, in castration, in amputations, excepting the largest joints, in removing tumors, as fatty tumors.

There are some operations in which it cannot be very well employed, especially in those about the perineum (hæmorrhoid operations, for instance), there being a great tendency for the patient to straighten the limbs out and interfere with the procedure.

Operations where very complete relaxation of the muscles is desirable are not well suited for gas anæsthesia.

Gas anæsthesia has certain disadvantages. It is expensive; this, however, could be overcome in a large hospital by manufacturing the gas in an apparatus in the operating room, as is done in their offices by some dentists who make a specialty of extracting teeth and employ large amounts of the agent.

The apparatus is a bit cumbersome to carry around in private operating, and still this is not a matter of much moment, as two or three cylinders and a gas bag and mouthpiece can easily be carried in a small dress suit case.

To one unaccustomed to the dark color of the patient's face and the dark blood in the wound, this method seems more dangerous than ether or chloroform anæsthesia.

The anæsthesia is not as profound as that of ether or chloroform, and the occasional talking of the patient may be disconcerting to one not familiar with the method.

In spite of these disadvantages, the great safety of the anæsthesia, the great rapidity of its action, the great comfort with which the patient can take it, the great freedom from nausea, the almost immediate recovery from the anæsthesia, the great freedom from lung complication, the

great freedom from kidney complication, the great freedom from extensive fatty degenerations of liver, kidney, and heart, which may follow chloroform anæsthesia and to a less degree ether anæsthesia—all of these combined make anæsthesia by nitrous oxid gas and air the method of choice in a considerable proportion of general surgical cases.]

CHAPTER IV

ACCIDENTS DURING ANÆSTHESIA, AND HOW TO MEET THEM

Every physician should have an intimate knowledge of the accidents which may occur during anæsthesia, and should be thoroughly conversant with the methods which should be employed to combat them. All these accidents are grouped under the term asphyxia, although this term, from the Greek σφύζειν, meaning to pulsate, refers only to the absence of the pulse. The discipline of the assistants must be perfect in order to prevent confusion and loss of time, and surgeons in charge should keep cool and collected. If an accident happens, the first thing that should be done is to remove the mask.

The accidents that may arise are *suffocation* and *respiratory* and *cardiac paralysis.*

Suffocation.—Suffocation is the result of mechanical interference with the air passages leading to partial or complete occlusion. It may be produced by mucus and saliva, which are secreted in large quantities when ether is given; by vomitus, which is expelled when the patient is awakening, or during anæsthesia when the anæsthetic is not given evenly, and in intestinal obstruction when enormous quantities are discharged. [An anæsthetist should always remember this danger, which occurs so often in patients suffering with ileus. Large quantities of vomitus are raised, and unless the anæsthetist is careful and the patient is watched until fully awake, the vomitus may collect in the pharynx and flow into the larynx, literally drowning the patient.] If there is any interference with the air passages the head should be immediately lowered and turned to the side, and a mouth gag inserted, in order to permit the mucus and saliva or vomitus to flow out. If the operation is not yet completed, the anæsthesia should be continued in this position, and, as a rule, the vomiting quickly subsides. Materials which collect in the recesses of the cheeks should be removed with steel sponges. If gurgling respiration and cyanosis indicate that mucus and saliva or vomitus has entered the larynx, a steel sponge should be passed into this organ and the material removed by a twisting motion. In desperate

cases a tracheotomy may have to be performed, and the aspirated material sucked out by a catheter, or something introduced to incite coughing.

A number of different methods may be employed to prevent blood flowing into the larynx during operations upon the jaws, cheeks, lips, nose, and floor of the mouth. This is a grave danger, as the aspiration of blood is frequently followed by broncho-pneumonia. This may be prevented by position, the head being allowed to hang over the end of the operating table, as advocated by Rose, or by performing a preliminary tracheotomy and inserting a tampon canula through which the anæsthetic may be given. The pharynx and aperture of the larynx may then be tamponed. [It should be remembered, however, that tracheotomy is rather a serious procedure, accompanied by fairly high mortality. This should not be employed when simpler methods will suffice.]

In operations upon the mouth the blood may be constantly wiped away with sponges on forceps, or the recesses of the cheek may be tamponed, while in operations upon the nose the posterior nares may be tamponed. In some cases it is well to induce only a partial anæsthesia; the reflexes are then preserved and the patient can expectorate the blood.

Foreign bodies, such as tobacco, artificial teeth, and candies, may pass into the air passages during anæsthesia and cause suffocation. Of course all foreign bodies should be removed before anæsthesia is begun, but if such an accident should happen, the mouth gag should be inserted and the foreign body removed with the finger or curved forceps.

The tongue may be spasmodically pressed against the pharynx during the stage of excitement, embarrassing respiration; usually this is associated with spasm of the diaphragm and the other muscles of respiration. The jaws should then be immediately opened and the tongue drawn forward with tongue forceps. If this does not suffice the hyoid bone should be elevated, using von Bergmann's method (*vide* p. 110) and artificial respiration begun.

During deep anæsthesia respiration may be embarrassed by the sucking in of the lips, cheeks, and alæ nasi. The lips are drawn in, especially in people without teeth and patients who have had a harelip repaired. It may also be embarrassed by a kinking of the trachea, when the head is in a poor position.

If the nasal passages are occluded by polypi, hypertrophied turbinate bones, tonsils, or a tampon, the jaws must be kept separated by a mouth gag and the tongue held forward.

The surgeon should be able to prevent deaths from suffocation occurring during anæsthesia.

Respiratory Paralysis.—Respiration may cease in the first stage of anæsthesia, the diaphragm being in the position of expiration, perhaps associated with a spasm of the glottis. This condition is apparently of

reflex origin, following stimulation of the trigeminal branches supplying the nasal mucous membrane, and occurs most frequently when large quantities of ether are administered quickly. The pulse remains good and the pupillary reflex is retained. If artificial respiration is performed quickly, the patient may vomit and then begin to breathe again.

If the anæsthetic is then forced because the patient vomits, this condition may occur again. These cases are known as "bad anæsthesias." If the condition occurs whenever the patient is anæsthetized, there is usually syphilis, tuberculosis, or some other disease of the nose, pharynx, or larynx. Painting of the mucous membranes with a five per cent solution of cocain before the anæsthesia is begun may be of value in these cases.

If the cessation of respiration occurring in the first stage of anæsthesia is overlooked and more anæsthetic administered, death may occur from cardiac paralysis, which is also probably of a reflex nature, following stimulation of the superior laryngeal nerve.

Cessation of respiration, occurring in deep anæsthesia, the result of administering too much anæsthetic, is much more dangerous than that above described. The pupils become dilated and do not react, and the heart stops beating after some seconds or minutes. If artificial respiration and heart massage are begun immediately, the pulse returns and then the respirations, but the patient remains for a long time in deep anæsthesia with contracted, fixed pupils.

Cardiac Paralysis.—The worst and most serious accident is that of sudden cessation of the heart beat, which may occur as the so-called early syncope, even in the first and second stages of anæsthesia. It is most frequent when chloroform is given, and is probably caused by paralysis of the cardiac centers or acute dilatation of the heart developing during the stage of excitement. It occurs most commonly in patients with some lesion of the myocardium, such as fatty degeneration so common in chronic alcoholics and following severe infectious disease; in patients with a chlorotic and lymphatic constitution; in anæmias following injury or internal hæmorrhages, leukæmia; in shock, and severe psychical excitement.

It may be caused during deep anæsthesia by a reflex paralysis following irritation of the sensory nerves (e. g., by traction upon the spermatic cord, by rough manipulation of the abdominal viscera, being analogous to shock induced by Goltz tapping experiments) and by the administration of too much anæsthetic, especially when there is an anæmia of the brain, the result of severe hæmorrhages or cardiac weakness.[1]

[1] The athetoid flexor movements of the fingers, which are regarded by Koblanck as a positive sign of approaching cardiac failure, are frequently present during perfectly normal anæsthesia, and the author does not attach much significance to them.

When these accidents occur the pulse becomes weak and irregular, the face pallid and corpselike, the pupils dilated and fixed. Irregular respiratory movements continue for some minutes after the heart stops beating.

Fortunately such accidents are but rarely seen when ether is used, being more frequent when chloroform is employed. If such an accident occurs the patient should be inverted immediately or, better, the foot of the table should be elevated to at least 45°, the object of the elevation being to overcome the cerebral anæmia and to favor the return of the venous blood which has accumulated in the splanchnic area to the right side of the heart. Artificial respiration, massage of the heart, transfusion of salt solution should also be employed. Action must be immediate. If after fifteen minutes there is no response to the treatment, the patient rarely recovers.

FIG. 65.—SHOWING INVERSION OF PATIENT AND METHOD OF PERFORMING ARTIFICIAL RESPIRATION SIMULTANEOUSLY. (Hare.) From Park's *Modern Surgery.*

The physician is not responsible for deaths from cardiac paralysis if the anæsthetic has been properly given, if there were proper indications for general anæsthesia (*vide* p. 117), and if effective measures were promptly instituted to relieve the conditions.

The purpose of artificial respiration is to carry oxygen to the blood, to favor the flow of oxygenated blood to the respiratory and cardiac centers, and to hasten the excretion of the anæsthetic from the lungs.

Freeing of Air Passages.—Naturally the air passages must be free before artificial respiration is begun. The mouth should be opened with a mouth gag, of which there are a number of different varieties. The Heister or the König-Roser should be inserted behind the back teeth on one side, after the jaw has been pushed forward; the von Bruns mouth gag if used should be applied between the incisor teeth. The index finger

may then be passed over the dorsum of the tongue and the epiglottis, until the easily palpable hyoid bone is reached, which should be drawn forward and upward. The tongue may be drawn forward more effectively by this procedure, introduced by von Bergmann, than by the use of tongue forceps.

Artificial Respiration.—Artificial respiration is usually performed according to the method introduced by Silvester.

The patient is placed in a horizontal or slightly inverted position; the operator stands behind him, grasps the arms flexed at the elbows, presses them against the sides of the chest, and then draws them backward until they are stretched horizontally above the head. By this procedure the ribs are raised by traction of the pectoral muscles and artificial inspiration is produced. When the arms are depressed expiration is produced. [Artificial respiration should never be performed more rapidly than the normal respiratory movements, eighteen to twenty complete movements being performed in a minute. If performed more rapidly and roughly, artificial respiration is apt to do about as much harm as good.] Care should be exercised not to fracture ribs, especially in old people with rigid thoracic walls.

FIG. 66.—SAME AS FIG. 65. From Park's *Modern Surgery.*

Cardiac Massage.—Heart massage, according to König and Maas, may be performed by the physician holding the tongue forward. He should stand upon the right side of the patient, using the left hand to hold the tongue, and should place the right hand flat upon the præcordial region, alternately raising and depressing the wrist joint and ball of the thumb, the number of complete movements corresponding to the number of heart beats.

Artificial respiration and heart massage should be continued until lungs and heart resume spontaneous activity, or if there is no reaction

for at least one hour. Intravenous infusion of physiological salt solution should always be employed with these methods.

Rhythmic Traction of the Tongue, Adrenalin, Faradism, Direct Massage of Heart, etc.—Besides these important measures above noted others may be mentioned, such as stimulation of the respiratory musculature by rhythmic traction of the tongue (Laborde), faradic stimulation of the phrenic nerves, injection of a few c.c. of a one per cent solution of adrenalin (Gottlieb, Mankowsky), the injection of a few c.c. of camphorated oil, and direct massage of the heart. The latter recommended by Prus for desperate cases, was first employed by Tuffier. It has not been successful, although in a number of cases the heart has been stimulated to beat for a short time (Zesas, Sick).

Death occurring during anæsthesia is generally ascribed to the effects of the anæsthetic. If it occurs, a statement should be prepared concerning the indications for the operation and general anæsthesia; the results of previous examinations, which should have excluded all conditions contraindicating the use of ether or chloroform, or only permit of their use in case of emergency; the operative technic, the accidents, and the methods employed to counteract them. This statement should be signed by all present and by the anæsthetist or operator who is directly responsible.

It is practically impossible to make a short synopsis of lines of treatment that should be instituted to meet the different accidents which may occur during anæsthesia, but the following suggestions may be made:

1. If the respiratory movements are spasmodic in character, with entrance of some air, there is marked cyanosis, the blood becomes dark, and respirations are embarrassed, but the pulse is still present, it is generally sufficient to open the mouth, draw the tongue forward, and clear the air passages. Prompt action, as a rule, removes the conditions.

2. If the respiratory movements have ceased, perhaps the result of reflex paralysis occurring at the beginning of anæsthesia, or anæmia of the brain, but the pulse is present and the pupils react, artificial respiration should be performed after the respiratory passages have been freed. Recovery with vomiting is, as a rule, rapid.

3. If the respiratory movements have ceased, the pulse is present but weak, the pupils dilated but do not react, anæsthesia having been carried beyond the normal point, the foot of the table should be elevated, artificial respiration and heart massage begun. In favorable cases the pupils contract, the pulse becomes better, spontaneous respirations return in at least ten minutes, and the patient remains for some time in the stage of deep anæsthesia with contracted, fixed pupils. In the worst cases the pulse does not return and death is the result.

4. If the pulse is lost, the respirations superficial or suspended, there is maximum dilatation of the pupils and corpselike pallor, the foot

of the table should be elevated, artificial respiration and heart massage performed, and transfusion of salt solution given. Patients in this condition rarely recover, even when correct treatment is instituted immediately.

CHAPTER V

DIFFERENT METHODS OF INDUCING ANÆSTHESIA. CHOICE OF METHODS

Anæsthesia by Sequence.—It is at times desirable and advantageous to change anæsthetics during anæsthesia. Anæsthesia may be started with chloroform or with some other anæsthetic, such as laughing gas or ethyl bromid, and continued with ether (Kocher); this method is to be especially recommended if during long operations the heart's action becomes weak or cardiac weakness is feared in anæmic and weak patients.

Some surgeons (Madelung, Kölliker) begin with ether and continue with chloroform, as the dangers of the latter, which are especially pronounced during the initial stage of anæsthesia, may be avoided in this way. The long stage of excitement accompanying ether anæsthesia is often dangerous to patients with heart lesions, and in these cases a few drops of chloroform administered in the beginning quiets the heart and induces anæsthesia, which should be continued with ether, rapidly.

Administration of Morphin before General Anæsthesia.—In some cases a small dose of morphin may be given to advantage before chloroform or ether anæsthesia is begun. One sixth to one quarter of a grain should then be injected subcutaneously from fifteen to thirty minutes before the anæsthesia is begun. It quiets the patient, and there is less reaction when the general anæsthesia is begun and less is required to maintain anæsthesia. Morphin combined with atropin is especially to be recommended when alcoholics are to be anæsthetized. It is frequently administered before ether is given as the atropin lessens the amount of secretion.

The patient under the action of morphin falls into a semistupor, and but little anæsthetic is required; even if conscious, patients experience but little pain. Often they react when spoken to loudly, and many operators attempt to maintain this condition during operations about the mouth, as the cough reflex is preserved and the blood may be prevented in this way from flowing back into the larynx. It is probably better, however, in this case not to give morphin, as it is then difficult to keep the patient in this condition, as he passes rapidly into the deeper stages of anæsthesia in which the reflexes are abolished. It is better to use chloroform and produce only a superficial anæsthesia.

Anæsthetic Mixtures.—Mixtures of different anæsthetics have been recommended and used. The Billroth mixture is composed of three parts of chloroform, one of ether, and one of alcohol; the so-called Vienna mixture contains one part of chloroform and three of ether; the Linhart mixture, one part of alcohol and four of chloroform. Schleich's general anæsthetic contains ether, chloroform, and petroleum-ether, and has a boiling point which corresponds to body temperature.

Braun has devised an apparatus for mixing ether and chloroform, by which the amount of each may be accurately measured, and mixtures of different composition made, by which each anæsthetic may be given separately or in sequence.

Scopolamin-Morphin Anæsthesia.—Scopolamin-morphin anæsthesia has many disadvantages and dangers. It has been employed quite extensively of late, but the results have not been such as to warrant recommendation. In the cases reported up to the present time the death rate has been higher than that following the use of chloroform and ether.

Both ether and chloroform have their advantages and disadvantages, their adherents and opponents. In America ether is used much more extensively than chloroform, and unless there is some positive contra-indication it should be employed.

There is a possibility of death occurring whenever a general anæsthetic is administered, but when it does occur it is often difficult to determine whether the anæsthetic was the direct cause of death or not. It may have been due to the inexperience or gross ignorance of the anæsthetist, but it should be remembered that fatal results have occurred even during minor operations when a general anæsthetic was not administered. Some of these deaths are probably due to fright, others to pulmonary embolism. The first time Simpson was about to use chloroform, the flask broke and the chloroform was spilled. The operation was begun without any anæsthetic, and the patient suddenly died. It is probable that if chloroform had been administered in this case, it would never have been tried again as a general anæsthetic.

Late Poisonous Effects of Anæsthetics.—A number of articles have appeared lately dealing with the late poisonous effects of anæsthetics. General anæsthetics, especially chloroform, produce changes in important viscera, resulting in metabolic changes which often prove fatal. The symptoms produced by these changes were grouped under different terms before their etiological and clinical significances were clearly recognized. The possibility of the late poisonous effects of anæsthesia developing should always be considered in determining the anæsthetic which should be used in each case.

Bevan and Favill, after the observation of a fatal case and a com-

parative study of a number of cases reported in the literature, come to the following conclusions:

1. Anæsthetics, especially chloroform (ether to a very limited degree), can produce a destructive effect on the cells of the liver and kidneys and on the muscle cells of the heart and other muscles, resulting in fatty degeneration and necrosis, very similar to the effects produced in phosphorus poisoning.

2. The constant and most important injury done is that to the liver.

3. This injury to the liver cells is in direct proportion to the amount of the anæsthetic employed and the length of the anæsthesia.

4. Certain individuals exhibit an idiosyncrasy or a susceptibility to this form of poisoning which it is difficult to explain.

5. There are certain predisposing causes which favor this destructive effect of chloroform, among which are: (*a*) age—the younger the patient the more susceptible; (*b*) causes which lower the general vitality of the individual and probably the vitality of the liver cells, such as diabetes, previous recent anæsthesias, infections by pus germs, diphtheria, intoxications from a dead fœtus in the uterus, a gangrenous mass in the abdominal cavity, etc.; (*c*) exhaustion due to hæmorrhage; (*d*) exhaustion due to starvation; (*e*) exhaustion due to wasting diseases, such as carcinoma; (*f*) lesions which have resulted in extensive fatty degenerations, such as occur in the limbs in infantile paralysis; (*g*) chronic diseases involving both liver and kidney, such as cirrhosis and nephritis.

6. As a result of this fatty degeneration and necrosis of the liver cells, toxins are produced either by the liver cells themselves or as a result of the failure of these cells to eliminate substances which under normal conditions they do, but which under these abnormal conditions they fail to do, and these substances, therefore, may accumulate and produce toxic effects.

7. These toxins produce a definite symptom-complex which makes its appearance from ten to one hundred and fifty hours after the anæsthesia. This symptom-complex consists of vomiting, restlessness, delirium, convulsions, coma, Cheyne-Stokes respiration, cyanosis, icterus in varying degree, and usually terminates in death.

8. It is probable that milder degrees of this poisoning are recovered from, and that the transient icterus noticed after chloroform anæsthesia without other evident cause is due to such poisoning, and many cases which exhibit restlessness, fright, mild delirium, drowsiness, etc., after anæsthesia may be due to the same cause.

9. This disease is an hepatic toxæmia; the toxins producing it, hepatic toxins; and possibly the previous condition making its development easily possible should be described as liver insufficiency. Just as we have for a long time recognized a condition, uræmia, in which we find

arising from a variety of noxious agents—anæsthetics, poison, infections, pregnancy, etc., affecting the secreting cells of the kidney and preventing their normal function—a pathologic condition, accompanied with a certain definite symptom-complex; so we must now, we believe, recognize a condition involving the liver which may be caused by a variety of noxious agents (anæsthetics, poisons, infections, pregnancy, etc.), affecting the secreting cells of the liver and preventing their normal function, a pathologic condition which we must describe as hepatic toxæmia, accompanied with a certain symptom-complex, and showing certain definite changes post mortem.

We believe that the condition of acute fatty degeneration of the liver with resulting hepatic toxæmia is as definite a pathologic entity as is acute pancreatitis with fat necrosis.

10. As by-products in this toxæmia, but not as the essential poisons, are found acetone, diacetic acid, and beta-oxybutyric acid in the blood and urine.

11. Post-mortem examination reveals fatty degeneration of the liver, fatty degeneration and mild degree of inflammation of the kidneys, and, in extreme cases, fatty degeneration of heart and other muscles. The lesion of the liver we believe to be the overshadowing and important one, and the one which is responsible for the symptoms and fatal result. The injury to the liver, in some cases, is so great as to result in practically a total destruction of the organ.

12. Somewhat similar hepatic toxæmias resulting from fatty degeneration of the liver cells occur in other conditions, and are accompanied by very similar symptoms. These occur in iodoform and phosphorus poisoning, diabetes, puerperal eclampsia, and acute yellow atrophy of the liver.

13. This fatty degeneration of the liver with hepatic toxæmia following anæsthesia is almost invariably due to chloroform in the fatal cases. Ether is seldom the cause of a death of this kind.

14. This serious and even fatal late effect of chloroform, which has heretofore not been generally recognized, must still further limit the use of this powerful and dangerous agent.

15. The possibility of the development of hepatic toxæmia makes chloroform distinctly contraindicated in those cases in which there exist the conditions which seem to favor its development—i. e., diabetes, sepsis, starvation, hæmorrhage, the presence of intoxication from dead material, the presence of fatty degenerations, as already cited, after infantile paralysis, and lesions of the liver. The susceptibility of children to this hepatic toxæmia must be recognized. That chloroform is capable of producing these serious late poisonous effects is a strong argument against its employment, and an argument in favor of the more general use of

ether; and yet we are confronted at times with the Charybdis of ether pneumonia on the one hand, and the Scylla of chloroform hepatic toxæmia on the other.

16. The recognition of this danger of hepatic toxæmia is a strong argument against the employment of chloroform for long anæsthesia, as it can be shown that a two-hour chloroform anæsthesia is almost invariably fatal to rabbits and guinea pigs, from fatty degeneration and necrosis of the liver cells; and a two-hour chloroform anæsthesia in man is an exceedingly dangerous thing.]

Mortality Following the Different Anæsthetics.—It is difficult to determine by statistical studies the value and safety of an anæsthetic, as the effects of operations, injury, and disease must also be taken into consideration. According to Gurlt's statistics (1890–97) one death occurred in 2,075 cases of chloroform and one death in 5,112 cases of ether anæsthesia. Williams's statistics, covering a period of ten years, show that one death occurred in 1,236 cases of chloroform, and one death in 4,860 cases of ether anæsthesia. The statistics collected by Julliard are as follows:

	Administrations	Deaths	Rate
Chloroform	524,507	161	1 in 3,258
Ether	314,738	21	1 in 14,987

Statistics appear to favor ether. König, however, has not seen a fatal result in 7,000 cases of chloroform anæsthesia, and in von Bergmann's clinic and polyclinic during an interval of twelve years, in which time about 8,000 chloroform anæsthesias were administered, the author saw but one fatal result.

Indications and Contraindications for Ether and Chloroform.—Each anæsthetic has its contraindications, one being less dangerous under certain conditions than the other.

Chloroform is to be avoided and ether used instead whenever there is a disturbance of cardiac function or demonstrable disease of heart muscle. It should never be forgotten, however, that ether, and particularly the lung complications which may follow its use, may be a source of danger if the heart is affected. Chloroform may be used in valvular disease without danger if compensation is good.

Chloroform is to be preferred to ether when the lungs are diseased or respiration interfered with, as the result of narrowing of the respiratory passages (tracheal stenosis, goiter, inflammatory œdema of the mucous membranes, etc.). These conditions also favor aspiration of saliva or vomitus. General anæsthesia is contraindicated: (1) In all conditions

in which both ether and chloroform are contraindicated; (2) if the patient is greatly excited before the operation; (3) in constitutional diseases, in which the bodily resistance is greatly reduced (diabetes, severe anæmias, leukæmia, obesity, status lymphaticus or thymicus, exophthalmic goiter); (4) in general weakness (syncope, shock, hæmorrhage, cachexia); and (5) advanced nephritis. Finally, anæsthetics should not be given, unless unavoidable, to women in the second half of pregnancy, as an abortion may result.

In all conditions in which general anæsthesia is associated with great danger, local anæsthesia should be used if possible. Death has resulted, however, from the use of local anæsthetics in excitable, weakened subjects. Von Eiselberg saw a fatal result following immediately a strumectomy which was done with Schleich's infiltration anæsthesia for the relief of Basedow's disease. Von Bergmann lost a diabetic patient while amputating a thigh under local anæsthesia, and the author has seen many patients collapse when the same method has been employed.

The physician must decide whether a local or general anæsthetic is indicated, and must choose the anæsthetic to be used in each individual case. If he has studied the case carefully, and has noted the indications and contraindications, he cannot be held responsible for any accidents which may occur.

LITERATURE.—*Blauel.* Ueber den Blutdruck während der Aether- und Chloroformnarkose. Beitr. z. klin. Chir., Bd. 31, 1901, p. 271.—*Bornträger.* Strafrechtl. Verantwortlichkeit d. Arztes bei Anwendung des Chloroforms. Berlin, 1891.—*Braun.* Ueber Mischnarkose u. deren Ration. Verwend. Chir.-Kongr. Verhandl., 1901, II, p. 136.—*P. Bruns.* Ein automat. Mundsperrer. Beitr. z. klin. Chir., Bd. 19, 1897, p. 253.—*Czempin.* Die Technik der Chloroformnarkose. Berlin, 1897.—*Dumont.* Handb. der allgem. und lokalen Anästhesie. Urban u. Schwarzenberg, 1903.—*Flatau.* Ueber Narkosenlähmungen. Zentr. f. Grenzgeb., 1901, p. 385.—*Gärtner.* Ueber einen neuen Apparat zur optischen Pulskontrolle in der Narkose. Zentralbl. f. Chir., 1903, No. 36.—*Hofmann.* Aethertropfnarkose. Deutsche Zeitschr. f. Chir., Bd. 65, 1903, p. 403.—*Kappeler.* Chloroformnarkose. Kochers Enzyklopädie, 1901;—Chloroformierung mit messbaren Chloroformluftmischungen. Chir.-Kongr. Verhandl., 1890, II, p. 79.—*Kionka.* Narkose, in Eulenburgs Realenzyklopädie, 1898.—*Koblanck.* Die Chloroform- u. Aethernarkose in der Praxis. Wiesbaden, 1902.—*Kocher.* Chirurg. Operationslehre. Jena, 1902.—*Kochmann.* Zur Frage der Morphium-Skopolamin-Narkose. Münch. med. Woch., 1905, No. 17.—*König.* Herzmassage. Chir.-Kongr. Verhandl., 1893, I, p. 21.—*Kraske.* Ueber künstl. Atmung und künstliche Herzbewegung. Chir.-Kongr. Verhandl., 1887, II, p. 279.—*Läseck, Rys, Zahradnický.* Skopolamin-Morphium-Narkose. Zentralbl. f. Chir., 1905, p. 611, 612.—*Lewin.* Note zur l'emploi du vinaigre contre les vomissements consécutifs à la chloroformisation. Revue de chirurgie, T. 15, 1895, p. 786.—*v. Mikulicz.* Ueber die Narkose. Deutsche Klinik, Bd. 8, 1901.—*Benno Müller.* Narkologie. Leipzig, 1903.—*Mulzer.* Sommer 1906 noch nicht erschienen.—*Nettel.* Ueber einen Fall von Thymustod bei Lokalanästhesie. Archiv f. klin. Chir., Bd. 73, 1904, p. 637.—*v. Niederhäusern.* Die Skopolamin-Morphium-Narkose. I.-D. Bern, 1905.—*Offergeld.* Experim. Beitrag zur toxischen Wirkung des Chloroforms auf die Nieren. Archiv f. klin. Chir., Bd. 75, 1905, p. 758.—

Schneiderlin. Die Skopolamin-(Hyoscin)-Morphium-Narkose. Münch. med. Woch., 1903, p. 371.—*P. Sick.* Zur operativen Herzmassage. Zentralbl. für Chir., 1903, p. 981.—*Zesas.* Ueber die Massage des freigelegten Herzens im Chloroformkollaps. Zentralbl. f. Chir., 1903, p. 588.

CHAPTER VI

LOCAL ANÆSTHESIA

Attempts to diminish locally the pain sense, so that painless operations might be performed, were made even in olden times. Constriction of the extremities or compression of large nerve trunks used in earlier times is no longer employed, because of the dangers of temporary or permanent paralysis.

Anæsthesia Induced by Freezing—Ether Spray and Ethyl Chlorid.—At the present time physical and chemical methods of different kinds are employed. Anæsthesia by freezing is a physical method which has come into more general use since the ether spray was introduced by Richardson in 1866. It reduces the temperature of the cutaneous area upon which it plays to five degrees above zero (F.), and the skin, which must be perfectly dry before the spray is applied, suddenly becomes white after a few minutes, the nerves lose their excitability and conductivity, and sensation is lost. Anæsthesia induced by the ether spray is superficial and lasts but a short time. When the area thaws out a burning sensation is experienced.

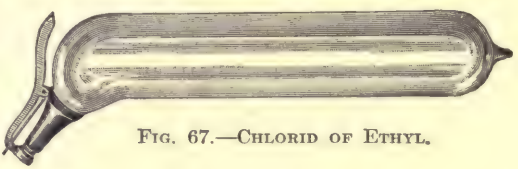


Fig. 67.—Chlorid of Ethyl.

Chlorid of ethyl, which may be bought in glass or metal flasks provided with detachable tops, is simpler in its application than the ether spray. It boils at 52° F., and when the flask is held in the hand a fine stream is discharged, which evaporates rapidly. The flask should be held from 30 to 40 cm. from the area to be operated upon, and as the temperature is rapidly reduced it is frozen and rendered anæsthetic more quickly than when the ether spray is employed.

Anæsthesia by freezing is suited only for small superficial incisions or for the introduction of an aspirating needle, and is to be especially recommended for incisions into circumscribed cutaneous and subcutaneous inflammatory processes.

Cocain Hydrochlorate.—Cocain hydrochlorate is the most important chemical agent for producing local anæsthesia. It was introduced into ophthalmology by Koller in 1884, and has become indispensable as a

local anæsthetic. It may be used in different ways in producing anæsthesia. Mucous membranes may be painted or sprayed with a solution of it; the drug may be injected around or directly into nerves, into the tissues, or subdural space of the cord.

Anæsthesia by Spraying or Painting with Cocain Solutions.—The mucous membranes of the mouth, nose, pharynx, and larynx may be rendered anæsthetic rapidly by spraying or painting them with a five or ten per cent solution of cocain. If inflamed the swelling rapidly diminishes as the vessels contract. Hollow organs such as the bladder may be rendered anæsthetic by irrigating them with a one per cent solution. The conjunctiva may be rendered anæsthetic by dropping a few drops of a one per cent solution upon the cornea. Only the surfaces of mucous membranes are anæsthetized in this way, but a number of operations not involving deeper tissues, such as the removal of nasal polyps and small superficial tumors and the opening of abscesses, may be performed.

Infiltration Anæsthesia.—Infiltration anæsthesia is employed very extensively in minor surgery. Schleich has done more by experimental work than anyone else to popularize and extend the usefulness of this form of local anæsthesia. Infiltration anæsthesia consists in the injection of weak solutions into the tissues. Weak solutions of cocain are much preferred to the stronger solutions, as the former are more reliable, and larger quantities may be used without the fear of toxic symptoms.

Schleich has three solutions of the following compositions:

Solution No. 1—Strong

Cocain hydrochlorate	gr. 3
Morphin hydrochlorate	gr. $\frac{2}{5}$
Chlorid of sodium	gr. 3
Distilled sterilized water	3 $3\frac{2}{5}$

Solution No. 2—Normal

Cocain hydrochlorate	gr. $1\frac{1}{2}$
Morphin hydrochlorate	gr. $\frac{2}{5}$
Chlorid of sodium	gr. 3
Distilled sterilized water	3 $3\frac{2}{5}$

Solution No. 3—Weak

Cocain hydrochlorate	gr. $\frac{1}{6}$
Morphin hydrochlorate	gr. $\frac{2}{5}$
Chlorid of sodium	gr. 3
Distilled sterilized water	3 $3\frac{2}{5}$

Schleich's tablets may be bought already prepared, and when the solution is required, the number of tablets required to make a solution of a certain strength should be added to a definite amount of water. Distilled sterilized water should be used for making the solution, as the cocain is decomposed and becomes ineffective when it is boiled.

Technic of Injecting Cocain Solution.—When the cocain solution is injected into the skin a definite technic must be employed. An ordinary hypodermic or larger syringe may be used for making the injection. The needle may be inserted at a point previously made anæsthetic by freezing,

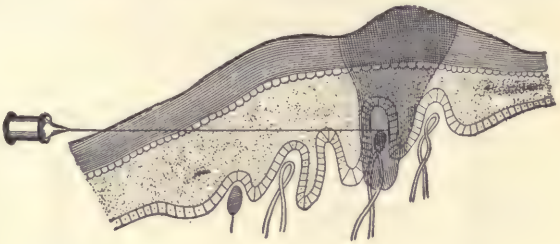


FIG. 69.—INFILTRATION OF DEEP LAYER OF SKIN.

and should then be passed almost parallel to the surface of the skin. The point of the needle should be kept in the cutis, and should not be passed into the subcutaneous tissues. If there is resistance to the piston when the injection is made, the needle is in the correct position.

The needle is then gradually inserted into the wheal previously raised until the entire line of incision is cocainized. Wherever the solution passes, a white hard wheal is raised. When the cutis is sufficiently infiltrated, some of the solution should be injected into the subcutaneous tissues. If it is the intention of the operator to carry the incision deeper the solution should be injected into fasciæ and muscle, and if the bone is to be exposed it should be injected into the periosteum.

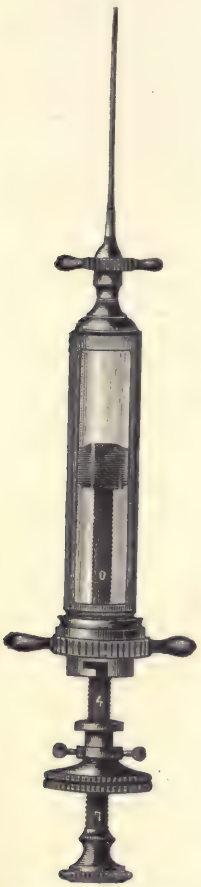


FIG. 68.—SYRINGE HOLDING 10 OR 15 C.C. WHICH MAY BE USED FOR INJECTING THE COCAIN SOLUTION.

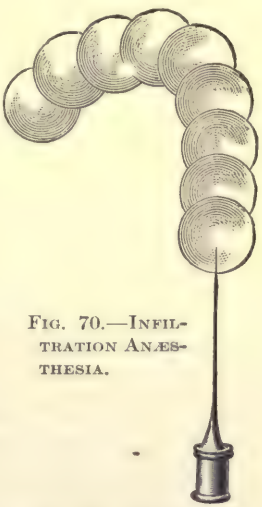


FIG. 70.—INFILTRATION ANÆSTHESIA.

If a large amount of cocain is used the injected area is transformed into a hard tumor-like infiltration which is anæsthetic.

The solution should be freshly prepared before being used. The needle should fit tightly and the barrel should be provided with a good handle, so that firm, even pressure may be exerted. A number of different apparatus have been devised for injection of the solution in which the air pressure in the flask containing the solution is raised by forcing in air with an ordinary syringe or bicycle pump. The solution is then slowly forced out of a rubber tube, to which a needle is attached, and the tissues are evenly infiltrated. [In the Bevan clinic a one tenth of one per cent solution of cocain is used for anæsthesia of the skin, and a one per cent solution for nerve blocking. The ordinary hypodermic syringe has been found very satisfactory for injecting the solution.]

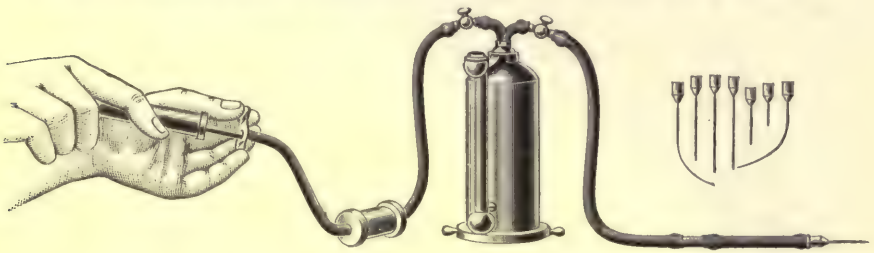


FIG. 71.—MATAS APPARATUS, INTRODUCING THE AIR.

The strongest Schleich solution has the following advantages: The injections are less painful, and as less of the solution is required a better view of the tissues may be had and their anatomical relations may be more easily recognized. It has the disadvantage that enough of the solution cannot be employed with safety to render anæsthetic large areas. When the weakest solution is employed the injection may be painful, and so much of the solution is required that the appearance of the tissues may be so changed and the anatomical relations so altered that it may be practically impossible to find small subcutaneous tumors, glands, or foreign bodies.

Infiltration anæsthesia may be employed when there is no acute inflammation. The injury of the tissues by the needle, and the danger of forcing bacteria and their toxins into healthy surrounding tissues, prevent its use in infections. Besides, in acute inflammation it may be necessary to make the incision layer by layer, and this is practically impossible when infiltration anæsthesia is used.

Many major operations (removal of large tumors, thyroidectomy, herniotomy, resection of ribs) may be performed under infiltration anæsthesia. Frequently, however, the patients become greatly excited, and general anæsthesia is to be preferred when there are no contraindications.

Anæsthesia by Nerve Blocking.—Anæsthesia by nerve blocking, in which the injections are made into the tissues surrounding the nerves or directly into the latter, has recently been improved by Braun and may be employed in a number of different ways.

According to Corning and Oberst, this method is especially suitable for producing anæsthesia of the fingers and toes. It should be employed as follows: The finger or toe to be anæsthetized is first rendered bloodless by applying a constrictor about its base. Then four subcutaneous injections of a one half or one per cent cocain solution are made about the base of the digit distalward to the constrictor until a circular swelling is raised by the solution. All the nerve connections are then blocked, the digit becomes anæsthetic in five minutes, and remains so until the constrictor is removed.

This procedure can only be employed for the incision of acute inflammatory processes, when the injection can be made into healthy tissues.

According to Hackenbruch, anæsthesia may be induced by the subcutaneous injections of a one per cent solution of cocain about the field of operation when only superficial operations are to be attempted.

Larger parts of the extremities, such as the hand and foot, forearm, and leg, may be rendered completely anæsthetic, according to Braun and others, if after the application of an elastic constrictor a one per cent solution of cocain is injected distalward to the constrictor about the larger nerve trunks (perineural injection). Subcutaneous injections of Schleich's solution may be combined with this method, the injections being made parallel to the extremity, completely or only partially about it, blocking effectually the cutaneous nerves.

The Use of Adrenalin in Cocain Solutions.—Braun discovered that the action of cocain and cocain solutions could be prolonged and increased about fourfold if, just before being used, a few drops of a 1:1,000 solution of adrenalin were added to them. It also renders unnecessary the troublesome and often painful elastic constriction without which anæsthesia cannot be successfully produced with one half to one per cent solutions. The vessels are constricted and an ischæmia produced by this agent, and therefore absorption is delayed. A complete anæsthesia develops in thirty minutes after perineural injections.

According to Braun, not more than five drops of the adrenalin solution should be used, and it should be added to the cocain solution just before the latter is injected.

For the perineural injection of the larger nerve trunks a syringeful of a one per cent solution of cocain or eucain, to which are added from one to three drops of adrenalin solution for each c.c.; for the injection of subcutaneous nerves along their course a one half per cent solution

with the addition of one drop of adrenalin solution for each 10 c.c. should be employed.

According to Braun, this procedure is best suited for producing an anæsthesia of the fingers, toes, hand, and foot, of the nerve trunks in the lower third of the forearm and leg, the ulnar nerve at the elbow, the peroneal and tibial nerves in the popliteal space, the long saphenous nerve, the superior clunial and supraclavicular nerves. The following nerves in the head and neck may be blocked by this method: the superficial cervical, the auricularis magnus, and the superior laryngeal, bilateral blocking of which produces a very satisfactory prolonged anæsthesia of the entire larynx, the supra- and infraorbital nerves, and the long cutaneous nerves of the scalp.

Spinal Anæsthesia.—[Lumbar or spinal anæsthesia was first employed by Corning; it was rediscovered and improved by Bier.] This form of anæsthesia is produced by injecting weak solutions of cocain or closely allied drugs into the lumbar meningeal sac.

The following technic is employed: The patient is either placed upon his side or seated upon a table with his back toward the operator, the body being somewhat flexed in order to separate the laminæ and render the intervertebral spaces wider. A thin canula 10 c.c. in length is then inserted between the spinous processes of the third and fourth lumbar vertebræ, just above a line uniting the highest points of the cristæ ilii (as in Quincke's lumbar puncture), or between the spines of the second and third lumbar vertebræ. This needle is then passed into the lumbar sac, and a few drops of cerebrospinal fluid are allowed to flow out, and then the solution is slowly injected. The solution mixes with the cerebrospinal fluid and acts upon the intradural nerve roots and trunks, especially upon sensory bundles lying in the posterior part of the cauda equina. Loss of sensation, often accompanied by some motor paralysis, occurs in from ten to fifteen minutes and lasts for different lengths of time, even up to two hours. The anæsthesia is most marked in the extremities, about the anus and the perineum, and is usually so complete that any operation may be performed below the level of the navel.

In many cases the anæsthesia is incomplete or does not develop; sometimes, when the canula has been directed lateralward between the nerves of the cauda equina, the anæsthesia occurs only upon one side (Dönitz).

Spinal anæsthesia as first employed was not practical. The mortality was high, and unpleasant symptoms (chills, sweating, nausea, vomiting, collapse) and after effects (headache, dizziness, vomiting, fever, paresis of the muscles of the extremities, and paralysis of the muscles of the eye) were frequent, especially so when the anæsthesia extended above the level of the navel.

Bier is of the opinion that the addition of adrenalin solution makes spinal anæsthesia practical. Adrenalin prevents the diffusion of the anæsthetic toward the brain, and diminishes, even if it does not prevent, the unpleasant symptoms and after effects. Care, however, should always be exercised; too large amounts of fluid and too strong solutions should not be injected; the injection should be made slowly; and elevation of the pelvis should be avoided (Braun). Elevation of the pelvis is of advantage when the anæsthesia does not extend upward high enough (Dönitz).

Stovain, with the addition of adrenalin solution, is, according to Bier, usually best suited for spinal anæsthesia, although unpleasant symptoms and collapse may develop when it is used. The solution may be bought sterilized and ready for use. It comes in small sealed glass bulbs, containing 2 c.c., in which are found 0.08 stovain, 0.0022 sodium chlorid, and 0.00026 adrenalin. Only half of this solution is used for an injection (therefore 0.04 of stovain).

All the chemical methods of producing anæsthesia are associated with danger, as the drugs which are used, especially cocain, are toxic.

Cocain Poisoning.—Acute cocain poisoning occurs most frequently when concentrated solutions are used, or when the solution is accidentally injected into a vein. The first danger may be avoided by using one per cent or weaker solutions; the latter by aspirating, when deep injections are made, to determine before the injection is made whether or not a vein has been entered. Never more than 0.1 of cocain should be injected when a one per cent solution is used, and never more than 0.15 when a one tenth per cent solution is used. In patients who are especially susceptible, toxic symptoms may develop after spraying or painting the mucous membranes with a five per cent solution.

The symptoms of cocain poisoning, which manifest themselves shortly after the injection or application of the solution, are anxiety, a sense of oppression, dizziness, collapse, convulsions, paralyses of different groups of muscles, and finally respiratory and cardiac paralysis ending in death. There is no chemical antidote to cocain poisoning. The patient should be placed in the horizontal position, with the lower extremities elevated to overcome the cerebral anæmia; coffee, cognac, and subcutaneous injections of camphor should be given. As soon as the respiratory movements become irregular, artificial respiration should be begun and continued until improvement is noted or the patient is past all hope of recovery.

A number of different substitutes for cocain (tropacocain, eucain, acoin, holocain, aneson, orthoform, nirvanin, anæsthesin, subcutin, stovain, alypin, novocain), which are less toxic but have about the same action, have been introduced. Tropacocain, eucain, and novocain have

about the same action as cocain, and have the advantages of being less toxic and of not being decomposed by boiling (Braun).

The cheaper preparations, eucain and novocain, are to be recommended for ordinary use in strengths of from 0.1 to 1 per cent, being prepared with physiological salt solution.

There are a number of important points to be considered when deciding whether a general or a local anæsthetic should be administered.

If the operation can easily be performed under local anæsthesia, it should be recommended to the patient. Then, if he desires a general anæsthetic, the surgeon should not persist in giving a local one, as the excitement accompanying this form of anæsthesia is frequently associated with grave dangers (fatal collapse).

It is better to employ general anæsthesia in the performance of major operations, even when local anæsthesia is technically possible. Under local anæsthesia the time required for the performance of the operation is lengthened, and the dangers of wound infections are increased. The fear which many patients have of a major operation and the attention which they pay to the details of the same may cause dangerous syncope. If the anæsthesia is not complete, the patient often complains bitterly, and the operative skill and technic of the surgeon suffers.

If there are contraindications to general anæsthesia, local anæsthesia or lumbar anæsthesia should be attempted even when major operations are to be performed. If the anæsthesia is not a success or the patient becomes greatly excited, general anæsthesia should then be carefully induced.

Spinal anæsthesia is to be attempted in operations below the level of the navel, when general anæsthesia is contraindicated, and the operation is to be extensive and upon a part where local anæsthesia is impossible. Spinal anæsthesia is especially suited for operations upon weak old people, and for amputations for diabetic and senile gangrene.

In studying the after effects of major operations performed under local anæsthesia, von Mikuliez has made the important observation that pneumonia following laparotomies, herniotomies, and thyroidectomies, are about as frequent after local as after chloroform anæsthesia.

LITERATURE.—*Bier.* Ueber den jetzigen Stand der Rückenmarksanästhesie, ihre Berechtigung, ihre Vorteile und Nachteile gegenüber anderen Anästhesierungsmethoden. Chir.-Kongr. Verhandl., 1905, II, p. 115.—*Braun.* Die Lokalanästhesie, ihre wissenschaftlichen Grundlagen und praktische Bedeutung. Leipzig, 1905;—Ueber einige neue örtliche Anästhetica. (Stovain, Alypin, Novokain.) Deutsche med. Wochenschr., 1905, p. 1667.—*Dönitz.* Technik, Wirkung u. spezielle Indikation der Rückenmarksanästhesie. Chir.-Kongr. Verhandl., 1905, II, p. 527.—*Dumont.* Handb. der allgem. und lokalen Anästhesie, 1903.—*Reclus.* La cocaine en chirurgie. Paris, 1895.—*Schleich.* Schmerzlose Operationen. Berlin, 1894;—Ueber lokale Anästhesie. Die deutsche Klinik, Bd. 8, 1901, p. 37.

IV. GENERAL DISCUSSION OF PLASTIC OPERATIONS

CHAPTER I

DIFFERENT PLASTIC PROCEDURES

THE procedures included under plastic surgery are intended to cover or repair deformities the result of congenital defects, disease, or accident by the use of living tissue. For hundreds of years surgery has found in the repair of defects of the face by plastic operations one of its principal and most satisfactory tasks. A number of procedures have been introduced and methods attempted to restore natural form to parts of the face which have been partially destroyed or improperly formed. It would be impossible in a book of this character to discuss all the different varieties of plastic operations, their good and bad results, and how they have been modified to meet conditions arising during the course of an operation. In all these operations with their modification there is a general principle which makes it possible to group the different procedures, which, as a matter of fact, are only varieties of the principal type. The following division is probably the most useful:

1. **Approximation of the edges of a wound by "undercutting"** them, using, if necessary, lateral liberating incisions. This method, devised by Celsus, the father of plastic surgery, may be modified in a number of ways in closing small defects. "Undercutting" the edges of the

FIG. 72.

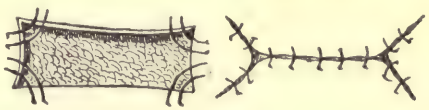


FIG. 73.

wound alone often permits of complete closure of small oval, elliptical, and rhomboidal defects (Fig. 72).

The greater part of a rectangular wound may be closed in this way, if the angles of the wound are first sutured and the long sides then united (Fig. 73). Lateral liberating incisions may be made to permit of approximation of the edges of the wound, the liberating incision being

allowed to heal by the formation of granulation tissue, or being sutured in the form of an angular incision (>) or a (Y).

In the Celsus procedure proper the defect should always be quadrangular in shape. The edges of the wound are mobilized by making

FIG. 74.

lateral parallel incisions passing out from the angles. If these incisions do not permit of approximation, a semilunar incision may be made on each side at some distance from the ends of the lateral liberating incisions. The semilunar incisions should only extend through the cutis,

FIG. 75.

and the concavity of the incision should be directed toward the wound (Fig. 75).

It can be seen from the examples already cited that this principle may be employed in a number of different ways in closing differently shaped defects: for example, in closing a quadrangular defect lateral

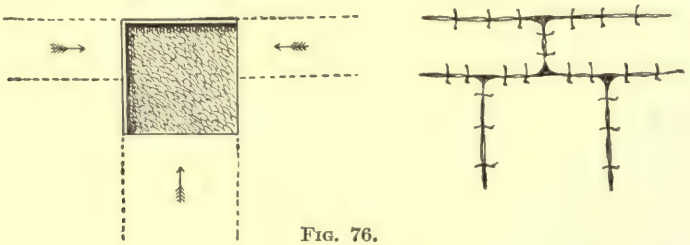


FIG. 76.

liberating incisions may be made upon only one, upon two, or even three sides. The defect should be thought of as composed of three right angles, each of which is to be covered by a mobilized flap. In this case two of the liberating incisions pass from the middle of the edge of the defect as represented in Figure 76.

In triangular defects the liberating incisions are made along the line of the base of the triangle to one or both sides (Fig. 77). If necessary, incisions as represented in Figures 74 and 75 may also be employed.

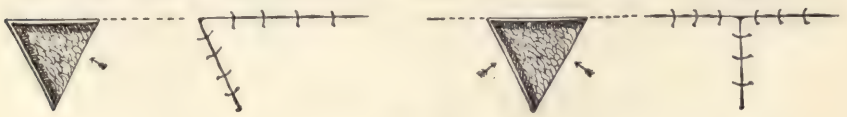


Fig. 77.

As a rule, the edges of the defect can be approximated much more easily if one or both of the lateral incisions are curved (Fig. 78).

Burow's method of closure of triangular defects is very ingenious, but is little employed at the present time. It consists in making lateral

Fig. 78.

incisions, each equal in length to at least two thirds of the width of the portion of the triangle to which they correspond. The flaps are then dissected up and approximated as indicated by the arrows in the accom-

Fig. 79.

panying figures. The shape of the wounds after suture is represented at the right hand side of the figures, showing shape of defect (Fig. 79).

In narrow rectangular defects a small triangular piece of skin can be excised upon the short sides to permit of linear closure (Fig. 80 *a*),

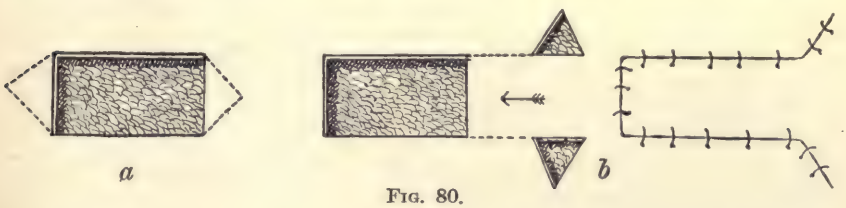


Fig. 80.

or the lateral liberating incisions of Celsus may be combined with Burow's method as represented in Figure 80 *b*.

In closing elliptical defects an incision may be made from the middle of one edge of defect (Lisfranc) (Fig. 81 *a*), and, if necessary, curved

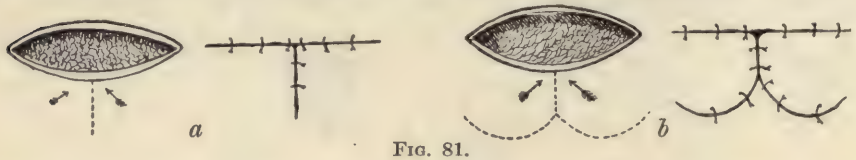


FIG. 81.

incisions may be made upon both sides from the extremity of this incision (Fig. 81 *b*).

2. **The Covering of the Defect by Flaps taken from an Adjacent Area.**—There are three different methods:

a. The lateral displacement of the flap (Dieffenbach's method).

b. "Jumping a flap," or torsion of the flap (Indian method).

c. Inversion and eversion of the flap.

a. The *lateral displacement* of a flap taken from the area immediately adjacent to the defect was devised by Dieffenbach, and was first practiced by him in Lisfranc's clinic in Paris in 1834 in the formation of an eyelid (blepharoplasty).

The defect should be triangular in shape, and in repairing the eyelids or lips the base of the triangle should correspond to the palpebral

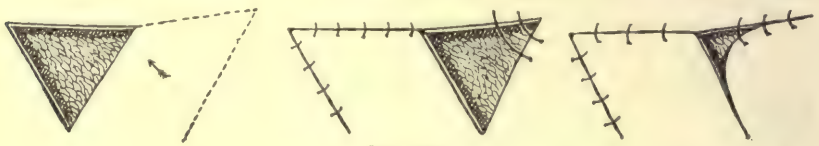


FIG. 82.

and interlabial fissures respectively. Upon one side of the defect a rectangular or rhomboidal flap is cut from the adjacent skin and dissected free from the subjacent tissues. The flap is then displaced, the corresponding sides of the defect and flap being sutured, while the side corresponding to the apex is not cut and forms the bridge or pedicle for the flap (Fig. 82).

FIG. 83.

After the flap is displaced, a triangular defect remains, which may be diminished in size by approximating the angles as represented in Figure 83. Double flaps may also be employed in Dieffenbach's procedure, then

the small lateral triangular defects remaining after displacement of the flaps must be allowed to heal by granulation tissue.

Elliptical defects may be closed by making curved incisions upon one or both sides, and forming flaps which may be displaced into the defect (Fig. 84 *a* after Hasner, and 84 *b* after O. Weber).

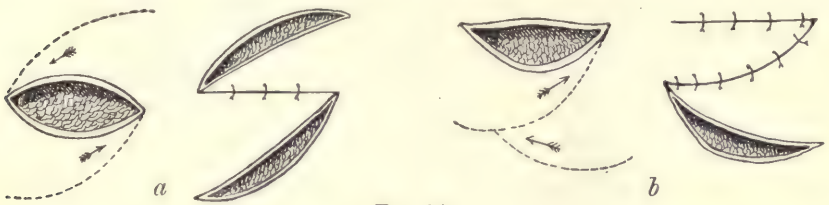


FIG. 84.

Flaps which are dissected free from the subjacent tissues, but still remain attached to the surrounding tissues at one or both extremities, are called pedunculated flaps.

b. The "*jumping*" or *torsion* of a flap corresponds to the old Indian methods in which pedunculated flaps from the cheek or forehead were used to repair nasal deformities. This so-called Indian method first became widely known through the writings of Carpue, an Englishman, in 1816. After it had been improved by von Graefe, it was perfected and extensively employed by Dieffenbach and von Langenbeck.

In this procedure pedunculated flaps of different forms are made from the adjacent skin, the pedicle of the flap being the only part of it which borders immediately upon the defect. In order to cover the defect the pedicle must be more or less twisted. (If the flap be not moved more than a quarter of a circle, twisting of the pedicle is not necessary, Bryant.) The form of the flap should correspond approximately to the form of the defect. The wound resulting from the separation of the flap may be diminished in size by suturing, or closed by other procedures.

According to Dieffenbach, one extremity of the incision made in forming the flap should extend into the defect, as the flap is mobilized better in this way, and then the pedicle lies directly upon the defect, and is not separated from it by a piece of intact skin (Fig. 85). The other extremity of the incision should be curved somewhat outward from the pedicle, rendering torsion of the flap without tension possible. Von Langenbeck laid great stress upon this point in the technic.

If the inner surface of the flap should be covered with epithelium, as in cheiloplasty, the flap should be folded upon itself and the wound surfaces held in approximation by sutures.

c. The *inversion* or *eversion* of a flap taken from the tissues imme-

diately adjacent to the defect relates to the employment of skin in the repair of mucous membranes. For example, a flap may be taken from the neck, and the surface of the flap turned toward the mouth cavity

FIG. 85.—DISPLACEMENT OF PEDUNCULATED FLAP UPON PEDICLE.

to take the place of the mucous membrane, the cut surface of the flap being covered with Thiersch grafts. The defects may also be repaired by folding pedunculated flaps upon themselves, one skin surface being turned into the defect.

3. **The covering of a defect by pedunculated flaps taken from distant parts** has its oldest prototype in the Italian procedure. It, like the Indian method, was employed at first only for rhinoplasty. This method first became well known through the writings of Taliacotius or Tagliacozza (1597).

The operation was perfected especially by von Graefe, and therefore it was known for some time as the German method. Flaps of different shapes are taken from the arm, forearm, or hand, and are sutured into the defects, the parts being immobilized until healing is complete. The pedicle of the flap may be cut, as a rule, in about ten days.

In this way defects of the face may be repaired with flaps from the arm, of the hand and arm with flaps from the breast, of the foot and leg with flaps from the healthy extremity.

4. **The covering of defects with nonpedunculated flaps taken from distant parts,** *grafting* or *transplantation* of skin, was probably attempted by the ancient Indians. In spite of repeated unsuccessful attempts, it was tried again and again. Reverdin's work, 1869, in which he transplanted small pieces of skin from 2 to 6 qmm. in diameter upon granulating surfaces, prepared the way for later studies and skin grafting as it is practiced to-day. Thiersch in 1886 perfected the method, obtaining permanent healing by transplanting upon surfaces from which the granulation tissue had been curetted away large broad strips of epi-

dermis containing the stratum papillare. This method, because of its simplicity and safety, is very extensively employed. The transplantation of cutis strips or flaps was promoted by the successful results obtained by von Buenger (1823), Wolfe, von Zehender, and von Langenbeck, and perfected by von Esmarch and Krause.

CHAPTER II

FUNDAMENTAL RULES FOR PLASTIC OPERATIONS

These rules have naturally undergone many changes in the course of time. The method of treating wounds at the present time has simplified these operations and increased their safety. It is most important that the operation be performed aseptically and that hæmorrhage be controlled, as the healing of the wound surfaces will then not be disturbed by suppuration or by the accumulation of blood. Care must also be taken that the blood supply of the flap is good, as its circulation and nutrition may be easily interfered with in freeing it, in forming and twisting the pedicle, in suturing, and in applying dressings.

Method of Separating the Flap.—A flap which is to be displaced or twisted should be of the same thickness throughout. This requirement may be easily fulfilled in the frontal and the temporal regions, in the scalp, and in other parts of the body, with resistant fascia and a thin layer of fat. It is more difficult, on the other hand, to make a flap of the same thickness throughout in the fatty region of the cheek, as the tissues cannot be made tense while it is being dissected free, and the flap is cut too thick in one part, too thin in another, and the nutrient vessels are sacrificed. When the incision has been carried down to the subcutaneous fat, the separation of the flap is begun opposite the pedicle. The incisions are made vertically to the subcutaneous tissues, separating the flap with a thin or thick layer of fat as far as the pedicle. In large flaps a thick layer of fat should be left attached to the skin, as it contains the blood vessels (von Langenbeck). The important requirement regarding blood supply may be fulfilled by making the length of the flap correspond to the course of the nutrient vessel—e. g., arteria temporalis, maxillaris externa. The pedicles of all flaps which are to be twisted should be made narrower than the body of the flap. The pedicle should, however, never be narrower than one half of the width of the flap. In old people it is better to make the pedicle too broad than too narrow. If the pedicle is not mobile enough to permit of torsion of the flap and

covering of the defect without tension, the extremity of the incision which does not end in the defect should be prolonged outward (Fig. 85). The pedicle should never be notched.

Form of the Flap.—The form of the flap should naturally correspond approximately to the form of the defect. Flaps with long, pointed extremities should be avoided, because of the danger of necrosis. If flaps are used which correspond exactly to the size of the defect, they will not be large enough, for the separated skin shrinks and tension will be required to approximate the edges of the defect and of the flap. Von Graefe made this mistake in his first attempts at rhinoplasty. Therefore the flap must always be broader and longer than the defect. The length of the flap may be measured by the fingers, the part forming the pedicle being the point from which the measurement is made.

Sutures to be Employed and Method of Inserting.—The sutures which unite the borders of the defect and flap should never exert any tension. Fine silk (preferably horsehair) should be used, and the edges of the skin should be approximated so that the resulting scar will be fine. The sutures should be inserted close to the edge of the skin, for sutures which include much tissue interfere with the nutrition of the edges, particularly of the end of the flap.

Methods of Applying Bandages.—Poorly applied bandages, producing pressure and constriction, also endanger the nutrition of the flap. Only when the dressings are intended to force the skin into a cavity—e. g., after removal of contents of the orbit—should any pressure be exerted.

In plastic operations upon the face the dressings should be changed frequently, as the skin about the mouth and nose rapidly becomes soiled. If a dressing is applied over the eyes, it should be changed daily in order to prevent the accumulation of secretion in the space between the lids and the irritation of the conjunctiva. The deepest layer of gauze covering the line of suture may be allowed to remain when the dressings are removed if there is no infection.

Effects of Inflammation and Venous Stasis upon Flaps.—Mild inflammation does not interfere with the healing of pedunculated flaps. Early removal of the sutures from the suppurating stitch holes, opening of the line of suture, where the exudate has accumulated, prevents the extension of the inflammation. If a phlegmonous inflammation develops, the flap should be raised and the underlying recesses should be tamponed and drained.

Frequently flaps become swollen and cyanotic after they are attached, and the epidermis may even be separated by a sero-hæmorrhagic exudate. These changes are due to passive congestion, and if the flap is punctured in many places the condition rapidly subsides in most cases. If the flap becomes dark blue or black in color it cannot be saved, and then moist

dressings (without rubber tissue) should be used to hasten the separation of the dead tissue. When the line of demarcation is sharply defined the necrotic tissue should be cut away.

Cutting of Pedicle.—The pedicle may be cut in from five to ten days, depending upon the size of the flap. In old people it should not be completely divided at one sitting, but should be divided at different sittings, intervals of two or three days intervening. The flap will be pale for a while after each incision. The edges of the divided pedicle should be freshened and should be sutured in position when divided.

Technic of Skin Grafting.—Skin flaps without pedicles demand a special technic, depending upon whether epidermal or cutis flaps are employed.

Epidermal strips (Thiersch) should be taken from the arm or thigh. While the hæmorrhage of the fresh wound or granulating surface, vivified by curetting or cutting away the granulations, is being controlled, the area from which the epidermis is to be taken should be washed and sterilized as in any other operation, and in addition washed off with a 0.9 per cent salt solution. The hæmorrhage from the fresh wound or granulating surface can be controlled most easily by compression with dry or moist gauze saturated with physiological salt solution or three per cent hydrogen peroxide solution. Only salt solution should come in contact with the epidermal strips; antiseptic solutions destroy the cells.

The skin should be put on stretch before the strips are cut. If the epidermis is taken, for example, from the inner side of the raised arm,

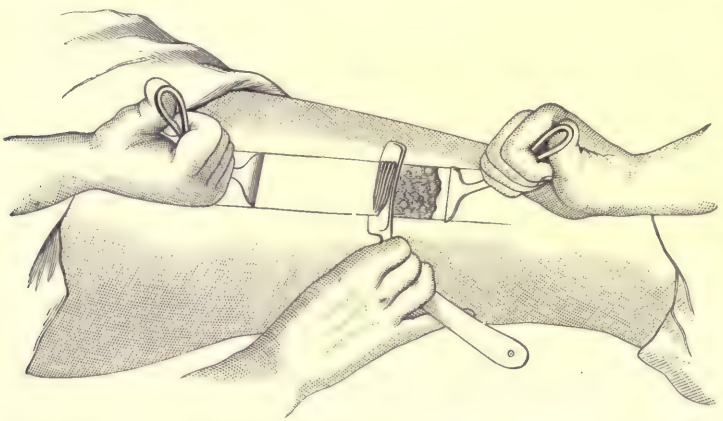
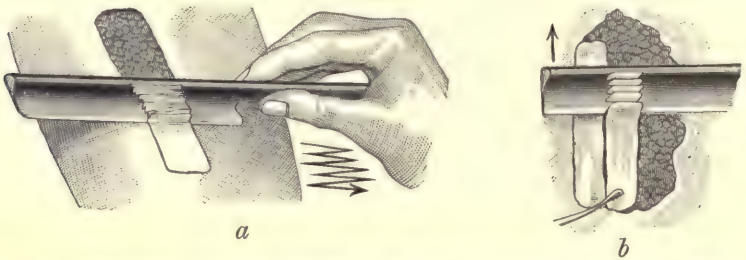


Fig. 86.—Cutting Grafts; Traction Hooks.

the assistant should stretch the skin toward the axillary fossa and the operator toward the elbow, the razor or knife being applied flat to the skin near the assistant's hand.

It makes little difference what kind of a knife is used, but it must cut well and be long enough. Broad and heavy knives with attached handles, razors ground on the flat (not hollow ground), or the ordinary



a *b*

FIG. 87.—*a*, CUTTING GRAFTS WITHOUT TRACTION HOOKS; *b*, PLACING GRAFT IN POSITION.

razor may be used. The author uses preferably a long, narrow, so-called phalanx knife.

The skin is then put on a stretch and the epidermal strips are cut with a sawing motion. The chief requirement is that the strip be of the same thickness throughout. If the strip is transparent, it is of the proper thickness, for then it must contain the stratum papillare with its germinal cells.

An experienced surgeon can easily cut strips 5 cm. wide and 20 cm. long, and the wound surface may then be evenly covered with skin grafts. The epidermal strips should then spread out upon the wound, which no longer bleeds, just as a large microscopic section is spread out upon a slide. The strips should extend about 1 cm. beyond the edges of the defect in order to become attached to and cover them. If a small amount of blood accumulates after the strips are applied it may be forced out by the slight pressure of scissors or other instruments. When the strips are placed upon a dry surface, agglutination by a thin layer of fibrin, which assists in rapid healing, occurs immediately (*vide* Wound Repair, p. 36).

Dressing of Skin Grafts.—A dry dressing is the best. A layer of iodoform gauze should be placed over the grafted area, and a few layers of sterile gauze over this. Adhesive plaster should then be applied to prevent displacement of the dressings. When the dressings are changed in from seven to ten days, unless there are indications for earlier removal, the lower layer of gauze should not be removed. It forms a firm crust with the wound secretion, which, however, permits of the escape of latter, under which healing occurs. After the healing is complete the gauze separates spontaneously. Ointments and moist compresses should not be used, as they macerate the epidermal strips. I can see no advantage in

the open treatment of grafted wounds which is recommended by Brüning. The latter believes that the rapid drying of the secretion following exposure to the air hastens agglutination.

If suppuration occurs, an attempt should be made to save as many of the grafts as possible. If the grafts are raised with tissue forceps, the underlying pus can be removed. Small white islands of epithelium are frequently found on granulating surfaces from which the epidermal strips have been separated by suppuration. If moist dressings which clean the granulation tissue are applied, and the excessive granulation tissue is destroyed by cauterization, these pieces of epithelium may proliferate and cover the defect. Von Mangoldt, guided by this experience, has used a simple method of skin grafting, which is of advantage in small granulating cavities. Minute pieces of epidermis are shaved off with a razor and placed upon the vivified granulating surface, after the hæmorrhage has been controlled (or upon healthy granulating surfaces), the epithelial cells being, as it were, assimilated.

The Thiersch method of skin grafting is often unsuccessful, because of the unhealthy condition of the granulating surfaces. Success should not be expected in granulating wounds following phlegmonous inflammations as long as they contain highly virulent pyogenic bacteria or the resistant bacillus pyocyaneus, as long as they secrete profusely, and appear unhealthy.

Schnitzler and Ewald have demonstrated that epidermal strips may heal upon nonvivified granulating surfaces, but the granulations must be clean and healthy.

In old people with flabby skin, or in small children, if no material is at hand, the epidermis from still-born children or amputated extremities may be used for grafting.

If a general anæsthetic is not required for some major operation, local anæsthesia may be used for skin grafting, subcutaneous injections in the course of the cutaneous nerves supplying the area, or circular injections, according to Braun, being employed. Infiltration anæsthesia is not suited for this purpose.

Cutis Strips.—In transplanting flaps comprising the entire thickness of the skin, the method described by Krause should be employed. Careful observance of asepsis and the dry operation are, according to Krause, the most important considerations next to the complete control of hæmorrhage. The hæmorrhage should be controlled by the ligation of the larger vessels, and by even compression with dry gauze. According to the experience of the author, the use of three per cent hydrogen peroxide solution for rapid control of the capillary hæmorrhage does not injure the tissues.

The elements of the transplanted cutis degenerate rapidly, and the

flaps must be taken with the greatest care to prevent more injury than that already produced by interference with its nutrition. Vigorous scrubbing, brushes and antiseptics are to be avoided in preparing the field of operation.

The strip of skin, in the form of an elongated spindle 6 by 20 cm., should be taken from the thigh or arm, in children from the back, and the resulting defect closed by sutures after the edges have been undercut. If, as frequently is the case in plastic operations upon the face (to cover simultaneously the cheek, temporal region, or adjacent parts of the neck), flaps the size of the hand and corresponding to the form of the defect are required, the resulting wound should be covered at once with epidermal strips.

In preparing the cutis strips, Krause directed all the incisions against the cutis, so that the strip when free contained no fat. Von Esmarch removed the skin and a layer of subcutaneous fat, and then spread it out upon the hand with the fat upward, and cut away the fat with a pair of curved scissors. Hirschberg has demonstrated that flaps containing subcutaneous fat will heal, but, as a rule, they have no advantage. In parts of the face where the least contraction of the flap produces distortion (about the eyelids) the author uses by preference flaps containing a thin layer of fat, as they become softer and more movable cutis than without fat.

In making a cutis flap it should be remembered that after it is cut it shrinks and becomes smaller.

If the wound to be grafted is not yet dry after the flap is cut, the latter should be folded so that the raw surfaces are in contact, further injury of the flap being prevented in this way. The flaps, when applied to the wound surface, become so tightly agglutinated by the fibrin layer that they appear to be glued together. Agglutination, which is prevented by the slightest hæmorrhage, is an important factor in the healing process. The edges of the defect and flap should be closely and accurately approximated. Sutures as a rule can be dispensed with, and as they exert tension, are not to be advised.

The dressings, as previously described in discussing the technic of epidermal grafting, may be employed.

The flap appears cyanotic after a few days, and its epidermis becomes separated. If some parts of the graft die, they dry under aseptic treatment, and are finally cast off by granulation tissue. If after ten days the greater part of the flap is reddish and warm, moist dressings may be applied to hasten the separation of the necrotic areas.

Inflammation prevents complete healing. If, however, the flaps have become firmly attached after a few days, they withstand severe inflammations, as no exudate is formed beneath them.

Use of Toes to Replace Fingers.—Concerning the healing of transplanted pieces of skin, *vide* Wound Healing.

Nicoladoni's procedure of using the tip of a toe with a nail to cover the bony stump of an index finger which had been partly torn away comes under the head of skin grafting.

Epidermal strips have the advantage of surety and rapidity of healing. Cutis strips, on the other hand, are more resistant, shrink less, and give better cosmetic results. On the face and parts of the body frequently exposed to trauma, such as the palm of the hand and the anterior surface of the leg, the cutis strips are to be preferred for grafting. Where the quick covering with skin of a granulating surface is desired, as after extensive burn, and where shrinkage of the grafted area is of no great significance, or it does not occur, as on the forehead, epidermal strips should be employed.

CHAPTER III

PLASTIC OPERATIONS WITH COMPOUND FLAPS, AND TRANSPLANTATION OF MUCOUS MEMBRANE, CARTILAGE, AND BONE

The pedicle which provides nourishment for and insures the viability of the pedunculated skin flaps permits also of the use of deeper lying tissues, such as mucous membrane, cartilage, and bone for the repair of defects.

In the Dieffenbach procedure (1834), employed in a number of different ways to repair the lips (cheiloplasty), the mucous membrane is transferred with the skin flap to form the red margin of the lip and its inner lining. If only a small area of mucous membrane is lost, a flap consisting of mucous membrane only is required.

Compound Flaps.—Compound flaps are used most frequently to repair bony and cartilaginous defects. The studies of von Langenbeck concerning the value of the periosteum for plastic purposes resulted in the introduction of his operation for the repair of cleft palate (uranoplasty, 1861), which is used as originally devised by him even at the present time. Two pedunculated flaps, consisting of mucous membrane and periosteum, are freed from the palatal processes of the maxillæ and united to cover the defect in the palate. The layer of bone formed in these cases is thin but sufficient. The skin-periosteal flaps taken from the forehead to repair nasal defects (von Langenbeck's periosteal rhinoplasty) are unreliable and insufficient as far as the formation of bone is concerned, and have been replaced by the skin-periosteal-osteal flaps. As early as

1855 Langenbeck had thought that it might be of advantage to remove a thin layer of bone with the skin and periosteum in performing rhinoplasty, but he desisted "because resection of the external table would open the veins of the diploë and phlebitis, and suppurative meningitis might follow this injury." König in 1866 was the first who attempted to perform a rhinoplasty of this kind. The attempt was successful, and the foundation for a great number of operations was laid. The skin-periosteal-osteal flap or cortical bony flap (König) is formed in the following way: When the skin is cut it is not raised from the subjacent periosteum, but a chisel is inserted and a thin plate of the external bony layer which remains attached to the periosteum is raised. The pedicle should contain no bone, as it would interfere with turning the flap. The entire flap, composed of skin, periosteum, and bone, is then twisted or displaced into the defect, which has previously been vivified; ossification occurs, and the defect is repaired. Skin sutures are sufficient to maintain the flap in position. If the piece of bone is separated from the periosteum by the chisel, it can be placed in the defect and covered with the flap of skin and periosteum (*vide* Transplantation of Bone).

This procedure has been used extensively in a number of ways: for rhinoplasty, for covering defects in the skull (König and W. Mueller), for filling in and repair of pseudarthroses (W. Mueller and von Eiselberg), and for the transplantation of small bones and of entire segments of bone.

It is possible, e. g., to use a pedunculated flap of the forearm, including a piece of the ulna, to form a nose according to the Italian method (Israel). The two distal phalanges of the finger may be replaced by those of the toe by a method introduced by Nicoladoni. In this operation a quadrilateral flap, the base of which is directed forward, is raised, and the extensor tendons and the joint (the interphalangeal or metacarpo-phalangeal of one or two toes depending upon the case) and the flexor tendon are divided. The skin upon the plantar surface remains attached and forms the pedicle. The toes are then attached to the vivified stump of the finger by skin, tendon, and bone sutures, and both extremities are immobilized in plaster of Paris dressings to prevent tension upon the pedicle, which is divided in about two weeks.

The following are examples of the different ways in which skin-periosteal-osteal flaps have been used in plastic surgery: Flaps from the sternum (Schimmelbusch) or clavicle (König) have been used to repair tracheal defects. Fritz König has used for the same purpose, with success, a compound flap, the cartilage being taken from the thyroid cartilage.

Osteoplastic operations resemble closely plastic operations with compound flaps. In these cases a layer of bone which retains its connection

with the soft parts is elevated, and after the completion of the operation it is replaced in the area which it formerly occupied (osteoplastic resection of the skull, Wagner; osteoplastic resection of the maxilla, von Langenbeck; of the nose, Ollier and von Bruns; the external wall of the orbit, Krönlein; the zygoma for different operations upon the trigeminal nerve). Sometimes parts of neighboring bones are retained in amputations and resections, and are displaced with the soft tissues attached to them to cover the ends of sawn bones (Pirogoff's osteoplastic exarticulation of the foot, using part of the os calcis to cover the sawn surfaces of the tibia and fibula; von Mikulicz-Wladimirow's resection of the foot, using the anterior part of the foot in a similar way; Gritti's amputation of the thigh, transplanting the sawn patella to the femur).

There are a number of different procedures which may be employed to repair small bony defects, being intermediate between the pedunculated skin-bone flaps and the direct transplantation of bone. In these a layer of bone is raised which remains connected with the periosteum, the latter forming the pedicle, about which the layer of bone is twisted (for repair of saddle-nose, amputation of the lower extremity by Bier's method to obtain a more useful stump).

The transplantation of mucous membrane is especially useful to replace the conjunctiva palpebræ in the formation of lids (blepharoplasty). The mucous membrane may be taken to the best advantage from the lips and cheeks. In blepharoplasty a pedunculated flap of skin is first transplanted, and after the flap has healed in position the mucous membrane is grafted (*vide* p. 47). The same technic is employed in grafting mucous membrane as in grafting skin.

Chips and plates of **bone** have been taken from the anterior surface or crest of the tibia to fill in a defect in the skull (Seydel), to fill in a sunken nasal bridge, and to repair a pseudarthrosis (Mangoldt).

The purposes for which cartilage may be transplanted have been previously mentioned.

Transplantation of bone has been employed extensively; results are more certain when fresh, living material is used. Bone chips and fragments, longitudinally divided long bones, or resected pieces of the latter may be used.

The metatarsal bones, the bones of the forearm and leg, have been longitudinally divided and used by Bardenheuer to repair defects in neighboring bones (Cramer).

Resected pieces of bone from the patient or from an extremity which has been recently amputated, may be used to repair a defect in another bone. Bergmann transplanted a piece of the fibula 12 cm. long into a defect in the tibia, following a resection for a sarcoma. The piece of the fibula was maintained in position by silver wire sutures. The soft tissues

must all be removed from the bone, which is to be employed for transplantation, and the latter must be thoroughly cleaned and boiled. Asepsis and the control of hæmorrhage are most essential factors in the success of bone transplantation. The transplanted bone should be maintained in position by sutures whenever displacement is likely to occur.

The advantages of the transplantation of bone as compared to the use of pedunculated skin-periosteal-osteal flaps are these: Larger fragments of bone may be used, and the large scars following the use of flaps —e. g., in the face—are avoided. If the transplantation is not successful, it may be tried again, or the more reliable skin-periosteal-osteal flap may be employed.

PART II

WOUND INFECTIONS AND SURGICAL INFECTIOUS DISEASES

I. THE NATURE OF INFECTION; THE LOCAL AND GENERAL REACTION

CHAPTER I

THE NATURE OF INFECTION

If materials from without which injure the tissues gain access to a wound, the latter is regarded as *contaminated* or *infected*. A wound infection may be caused by poisonous substances such as snake venom, as well as by bacteria, and therefore a purely toxic is differentiated from a purely bacterial infection. The bacterial are so much more frequent than the toxic infections that the term has practically been limited to the former, and when a wound, the field of operation, and the hands are spoken of as infected, the term is employed in this sense.

Toxic Wound Infections.—In toxic wound infections the poisons enter the circulation and produce a general toxic infection (e. g., snake venom).

Bacterial Wound Infections.—Bacterial wound infections may be followed by general infections, but these are not pure general bacterial infections, for toxins are produced by bacteria when they grow in the tissues, and are liberated when they die and are dissolved, and in these general infections the organism is flooded not only with the bacteria, but with their toxic products as well. Sometimes in the general bacterial infections the most marked symptoms are produced by the presence and multiplication of the bacteria in the blood stream and viscera, while in other cases they are due to the absorption of the toxins from the primary focus, few, if any, bacteria being found in the blood and viscera. Theoretically a general bacterial and a general toxic infection may be differentiated from each other, but practically it is not always possible to differentiate between the two. Tetanus, diphtheria, and many diseases caused by pyogenic and putrefactive bacteria are toxic infections, as the

action of the toxins elaborated by the bacteria found in these diseases gives to the latter their most essential characteristics. In most infections with pyogenic bacteria, in anthrax and plague infections, the micro-organisms invade and multiply in the blood and are deposited (metastases) in tissues (glanders, leprosy, miliary tuberculosis) producing the most prominent symptoms of the diseases.

The general putrefactive infections are frequently general toxic infections (sapræmia), developing secondary to putrefactive wounds from which the products elaborated by the putrefactive bacteria, only rarely the bacteria themselves, are absorbed.

Because of the similarity of the general clinical symptoms occurring in the general putrefactive, toxic, and bacterial infections, the term sepsis or septicæmia has been applied to all general infections caused by pyogenic bacteria. Bacteriologists, following Koch's example, regard any infection, regardless of the micro-organism (pyogenic cocci, plague bacilli, or the plasmodium of malaria), as septicæmia if the bacteria invade and multiply in the blood. [In the German edition of this book the terms sepsis, septicæmia, pyæmia, and a number of different terms which have been applied to mixed forms of general infection, are not used. Lexer describes a general pyogenic infection without metastases, a general pyogenic infection with metastases, and a general putrefactive infection. The words septicæmia, pyæmia, and sapræmia have become so well established in American medical literature that it seems best to use them in conjunction with the terms used by Lexer. To be sure, the terms have been used differently, and even at the present time are interpreted differently by different authorities. Pyæmia is synonymous with general pyogenic infections with metastases, septicæmia with general pyogenic infections without metastases, and sapræmia with general putrefactive infections.]

Monoinfections, Polyinfections, Reinfections.—Besides the simple infections (monoinfections), which are caused by one variety of micro-organism, there are the mixed infections (polyinfections) which are caused by two or more varieties of bacteria acting simultaneously. Later infection with another variety of micro-organism is called secondary or accessory infection. If infection occurs later with the same variety of micro-organism as produced the first infection it is called new or reinfection.

Causes of Inflammation.—From a surgical view point the most important causes of inflammation are of a plant nature; almost all belong to the class of bacteria. These are simple cells which multiply by fission, and for that reason are called fission-fungi or schizomycetes.

Action of Bacteria, Toxins, and Endotoxins.—Their action depends chiefly upon toxic materials, which are produced by them in the tis-

sues just as they are upon artificial media. The mechanical action of bacteria, such as is produced by the plugging of capillaries when large numbers circulate in the blood, is of much less moment than was formerly considered to be the case before we had accurate knowledge of the formation of toxins and the action of bacteria. Bacteria do not develop in large enough numbers to have any great mechanical action. No soluble toxin characteristic of the anthrax bacillus has been demonstrated, and it may be possible that in this infection the large number of bacilli circulating in the blood have a mechanical action in closing the capillaries. If so, this is the only infectious disease in which mechanical occlusion is the chief factor.

The poisonous materials produced by bacteria are in general of two kinds: sometimes one predominates, sometimes the other. These materials are produced by the action of the bacteria upon the tissues or upon culture fluids in which they are soluble, and are to be regarded as secretion products of bacteria. If a culture of bacteria which produces large amounts of poisons such as the tetanus or diphtheria bacillus is made, and the culture is then passed through a porcelain filter which is not permeable to the bacilli, the filtrate if injected into animals will produce the same symptoms as those produced by the bacteria, while the bacterial residue has no action. The filtrate therefore contains poisonous materials which have been produced in the culture media. These poisonous secretion products of bacteria are called toxins. Their chemical nature is not fully understood. Apparently they do not belong to the albumins proper, although they are closely related to them and to the ferments. They are extremely sensitive to chemical influences, particularly so to heat, and lose their toxic action immediately when heated to 80° C. and in a short time after being heated to 50° C. Their chief characteristic is their specificity, all toxins having a definite specific action corresponding to that of the bacteria by which they are secreted. The toxins of many bacteria (streptococci, staphylococci, bacillus pyocyaneus, bacillus of tetanus) contain bodies which dissolve (hæmolysins) or agglutinate (agglutinins) red corpuscles. Experiments have demonstrated that hæmolysis and agglutination are due to different substances.

The second variety of bacterial poisons are the bacterial protoplasmic poisons. They are the substances which are contained within the protoplasm of the bacteria, and are liberated only when the bacteria die and are dissolved. Buchner named the albuminous-like poisons which are obtained when cultures are boiled or ground up, bacterial proteins. Their action when the bacterial protoplasm contains no true toxin (endotoxin) is not specific. They have a common action which, as a rule, is pyogenic (Oppenheimer). The action of the proper pyogenic bacteria or microorganisms depends upon the toxins produced by them and endotoxins

which are freed when bacteriolysis occurs. The results of bacterial infection of a wound are twofold—local and general.

CHAPTER II

LOCAL REACTION

The local reaction begins with the entrance of the bacteria into the tissues (invasion). It does not, however, follow immediately the invasion, as the bacteria must first become adapted to their new surroundings, and must develop to such an extent that the bactericidal properties of the tissues can no longer restrain their growth. The length of the period of incubation varies, depending upon the number and virulence of bacteria and the resistance of the organism. It is of only a few hours' duration when the bacteria are derived from a patient with a severe infection (e. g., general pyogenic infection, meningitis, peritonitis, phlegmon). It is longer—according to Friedrichs's experiments at least six hours for the bacillus of malignant œdema—when, as is usually the case, the bacteria come from the outer world. Bacteria of a low grade of virulence frequently are unable to invade the tissues, as their growth is prevented by the bactericidal properties of the tissue fluids.

Bacterial toxins destroy the tissues, but they also irritate them and incite a number of processes which are intended to defend the tissues against their invasion. The more active the defense the more violent the local reaction. The usual local reaction may be absent if a weakened organism is attacked by highly virulent bacteria, as in such a case as this a powerful defense would not be possible. In experimental work there is no local reaction when the organism has previously been immunized against the bacteria in question; there being no necessity for defense, as the protective bodies which will not permit of the development and invasion of the bacteria are already present in large quantities.

The local reaction is inflammatory in character and varies in character and degree. It differs even with the same infection, and depends upon a number of factors, especially upon the virulence of the bacteria and the resistance of the tissues. In many cases the local reaction is characteristic, giving to the infection distinct clinical features. Streptococci from the same source may produce a serous, a fibrinous, or a suppurative inflammation. The local reaction produced by pyogenic cocci, diphtheria and tubercle bacilli is very different. In a purely toxic infection the general symptoms are produced by the absorption of the toxins, and the local changes have nothing characteristic—e. g., tetanus.

Inflammatory processes are characterized by three fundamental changes, notwithstanding their different clinical pictures. The changes are not only incited by bacterial toxins, but also by mechanical, thermal, and chemical irritation. [At the present time the changes produced by mechanical, thermal, and chemical irritation are regarded as reparative rather than as inflammatory.] The three processes are:

1. Disturbance of circulation with exudation.
2. Degenerative changes.
3. Regenerative changes.

1. **Disturbance of Circulation with Exudation.**—The vascular changes may be most easily followed when the mesentery of a frog or rabbit is spread out upon a glass slide and observed under a microscope. In this way the mesentery is exposed to the desiccating influence of the air and the irritating substances in it, and inflammatory processes are incited. The vascular changes begin with an active (congestive) hyperæmia, as the irritation paralyzes the vasoconstrictors and the vessel walls become

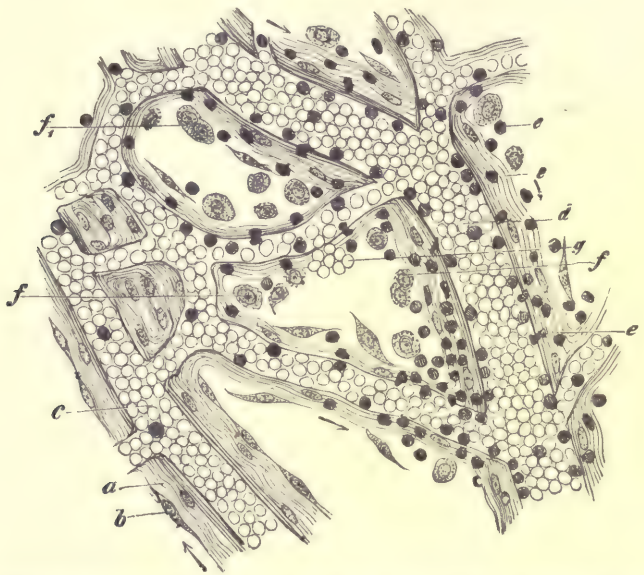


FIG. 88.—SECTION OF INFLAMED OMENTUM FROM MAN. (After Ziegler.) *a*, Normal trabeculæ in omentum; *b*, normal endothelium; *c*, small artery; *f*, detached endothelium; *f*, polynuclear cells; *g*, extravasated red blood corpuscles.

relaxed. The blood flows more rapidly through the arteries, capillaries, and veins. Soon, however, there is a marked slowing of the blood stream in the center of the inflammatory focus, and a passive hyperæmia succeeds the active, as the injured vessel walls offer more resistance, become

more permeable, and the surrounding tissues lose their tension. Often there is a transitory stagnation of the blood stream in the capillaries (stasis).

Migration of Leucocytes.—A peripheral stasis of the leucocytes in the veins, and their accumulation in the capillaries precedes the exudation. As soon as the blood stream begins to slow, the leucocytes pass to the peripheral portion of the blood current, which ordinarily is composed of plasma containing no cells. The heavier red blood corpuscles remain in the center of the stream. Slowly the leucocytes roll along the vessel wall, until single cells or whole groups of them become attached. This occurs much more readily in the capillaries, where there is often stagnation of the blood stream. Then follows the emigration of the leucocytes, which has been described by Cohnheim. Just as an oil drop swimming upon water changes its form when small parts are sent out, and assumes its form again when these flow back, so the motile protoplasm of the leucocytes sends out narrow pseudopodia through the vessel wall where the cement lines of the endothelium, which under normal conditions, and still more so when the vessel wall is injured, offer a point of exit. When the entire leucocyte has passed through the vessel wall, it begins to wander into the tissues.

Chemotaxis: Positive and Negative.—This active passage of the leucocyte from the vessel wall is due to the attractive action of the cause of the inflammation. This property of leucocytes and almost all motile cells of being attracted by definite chemical substances, particularly by most bacterial toxins and proteins, is called positive chemotaxis (*vide* Leucocytes, p. 159). Some bacteria, such as the bacillus of anthrax and malignant œdema, repel the leucocytes, this phenomenon being called negative chemotaxis.

Formation of the Exudate.—While leucocytes wander into the inflammatory focus from all sides and infiltrate the tissues, so that their structure can no longer be recognized, an exudate is being poured out from the vessels which separates the tissues and fills the tissue spaces. The pouring out of the exudate, which is to be regarded as a product of secretion rather than of filtration (Heidenhain), is due to an alteration of the vessel wall, in consequence of which the secretory function of the endothelium is altered. The injured vessel wall becomes more permeable than normal, so that materials which usually remain in the blood are no longer retained. The inflammatory exudate differs from lymph in that it contains greater numbers of cells and larger amounts of albumin. As a result of the exudate the hyperæmic area becomes tense and hard (inflammatory infiltrate) or presents the signs of œdema (inflammatory œdema), as it pits when pressure is made with the finger.

Important provisions for the protection of the tissues are combined

with these changes. In the non-bacterial inflammations the liquefaction of the necrotic tissue which renders absorption possible is the first protective step. The cellular exudate forms the first line of defense against bacteria, as it contains bactericidal bodies which are ably supported in their action by the leucocytes (phagocytes).

Polymorphonuclear Leucocytes, Plasma Cells, etc.—The cells which emigrate from the vessels are mostly polymorphonuclear leucocytes with neutrophile granules, which, according to Ehrlich, are derived chiefly from the bone marrow. If the vessel wall is severely damaged, red corpuscles which have escaped by diapedesis or rhexis, and other varieties of leucocytes, such as lymphocytes and eosinophilous leucocytes, the granules of which stain with acid dyes, are found. The lymphocytes are small, round, motile cells with large nuclei. They lie together in small groups (so-called small cell infiltration) and Ribbert is of the opinion that they form a small lymphatic focus, particularly in the neighborhood of the vessels, which increases as the inflammation progresses, and because they are intended to absorb noxious materials they remain for a long time.

2. **Degenerative Changes.**—The degeneration and necrosis within the inflammatory focus is partly due to the bacterial toxins, to mechanical, chemical, and thermal influences—all of which act upon the cells and frequently cause their death. They are also partly due to circulatory and nutritional disturbances resulting from the pressure of the exudate, the stasis in the capillaries, and the thrombosis of inflamed veins and arteries (*vide* Pyogenic Disease of the Vessels, p. 233).

The necrotic tissues become liquefied or absorbed by the leucocytes or the ferments liberated by them when they die. Liquefaction and absorption are rarely complete, and the necrotic tissue is only separated from the healthy, rarely completely liquefied or absorbed.

3. **Regenerative Changes.**—The first signs of regeneration are seen early (after twelve hours). The protoplasm of the connective tissue cells and of the endothelial cells of the lymph and blood vessels increases in amount, and numerous mitotic figures may be seen, indicating that the tissues are therefore proliferating actively. It is difficult to determine the origin of the large, round, mononuclear cells which stain heavily and characteristically with methylene blue. These have been called *plasma cells* by Unna, who regarded them as derivatives of connective tissue cells. According to Marschalko, Ribbert, and others, they are derived from large lymphocytes.

The growth of tissue increases as the hyperæmia and exudate subside, resulting in the development of granulation tissue, which is composed of fibroblasts, leucocytes, lymphocytes, newly formed and old ground substance, and many newly formed blood vessels. Eventually the granu-

lation tissue fills in the defects resulting from the necrosis and liquefaction of the tissues. Therefore granulation tissue is always found in ulcers of the skin and mucous membrane, about sequestra in bone, in the walls of abscesses and fistulæ, etc. The granulation tissue surrounding dead tissue aids in separating the latter (demarcation), and as the inflammation subsides, produces a secretion rich in leucocytes, the ferments of which digest the necrotic tissue.

The granulation tissue becomes transformed into scar tissue as wound repair progresses.

Symptoms of Acute Inflammation.—The classical symptoms of acute inflammation, which were described by Galen, are rubor (redness), tumor (swelling), calor (heat), and dolor (pain). The redness and the local elevation of temperature about the inflammatory focus are due to the active hyperæmia, the swelling to the exudate, the pain to the action of bacterial toxins, and the other harmful agents upon the nerve endings. The fifth symptom, which is sometimes described as functio læsa, the disturbed function of the part of the body involved, is due to the swelling and pain.

Character of the Inflammation and Clinical Course.—The character of the inflammation is determined by its cause. Sometimes all the symptoms are pronounced, sometimes they are but slightly developed; sometimes hyperæmia and exudation predominate (e. g., inflammations due to pyogenic bacteria); sometimes degeneration and necrosis (e. g., cauterization, burns, and frostbites of the third degree, in putrefactive and severe pyogenic infections); and sometimes the proliferation of tissues (e. g., in syphilis, actinomycosis, and some forms of tuberculosis).

The inflammations associated with hyperæmia and exudation have an acute clinical course, while those resulting in the formation of large amounts of new tissue develop gradually and are chronic. Intermediate forms are called subacute. The clinical course depends upon the virulence of the bacteria and the resistance of the tissues. An acute inflammation may become chronic, if the virulence of the bacteria diminishes; the reverse may happen if the virulence increases. The character of the exudate and the amount of local tissue degeneration characterize different forms of inflammation. The following forms may be distinguished:

1. In *serous inflammation* a watery exudate containing large amounts of albumin and but relatively few cells is formed. It gives rise in mucous membranes to a serous discharge mixed with mucus; in the cutis to the formation of vesicles with clear contents; in the subcutaneous tissue to an inflammatory œdema; in the large body cavities, joints, and bursæ to characteristic serous exudates. Serous inflammation may be acute (erysipelas), or chronic (tuberculosis of serous cavities), depending upon the bacteria. Serous inflammations may be associated with the mildest

(osteomyelitis albuminosa) or the severest infections caused by pyogenic cocci (seropurulent phlegmon). Frequently there are transitions to the fibrinous or purulent types, the fibrin is precipitated or the serous exudate is rich in cells.

2. *Fibrinous inflammation* is characterized by the precipitation of albuminous bodies dissolved in the exudate. Ferments which are apparently derived from degenerating cells, especially the leucocytes, are required for the precipitation of these bodies. The fibrin is deposited especially upon the surfaces of mucous, serous, and synovial membranes, upon ulcers of the skin, and in the alveoli of the lung, rarely in the connective tissues. The formation of fibrin is sometimes combined with necrosis (coagulation necrosis), sometimes with serous or purulent exudates. The fibrin forms upon the mucous membranes a gray or yellowish-white membrane—the fibrinous or croupous pseudo-membrane—which occurs in diphtheria and other inflammations of mucous membranes produced by the staphylococcus, streptococcus, pneumococcus, and typhoid bacillus. The fibrin layer separates easily, where only the underlying epithelium is destroyed, and for this reason in diphtheria of the bronchi branched fibrinous masses, casts of the respiratory passages, are coughed up. When the fibrinous threads are closely attached to deep necrotic tissue of the mucous membranes, the pseudo-membrane becomes firmly adherent (pharyngeal diphtheria).

Upon the serous membranes of the body cavities and the synovial membranes of joints and bursæ fibrinous deposits are at first loose, but become organized later to form villous or nodular growths or stratified callosities. In tuberculosis of joints and bursæ these fibrinous deposits are frequently torn loose by movements and rounded off to form bodies which resemble seed corn (rice bodies). A fibrinous membrane forms upon granulating ulcers of the skin in wound diphtheria, and is associated with purulent secretion in many different kinds of infections. Free flakes of fibrin in serous or seropurulent exudates are usually indicative of tuberculous inflammation.

Microscopically the fibrinous deposit or membrane is composed of an entangled network of threads of fibrin in which are inclosed epithelial and connective tissue cells, leucocytes, and groups of bacteria.

3. In *purulent inflammations* a yellowish, creamy exudate, the well-known pus, is formed. Pus occurs as a secretion of the mucous membrane mixed with mucus, as the contents of pustules, as an exudate in the tissues, as the contents of tissue spaces following liquefaction of dead tissues, as an exudate in serous cavities and joints (empyema). The characteristics of a purulent exudate are due to the large number of cells, especially the polynuclear neutrophiles, the so-called pus corpuscles, which it contains.

The longer the pus remains in the body, the greater the amount of necrotic tissue which is held in solution by the ferments liberated by the degenerating leucocytes it contains (Fredr. Mueller). Old pus contains fresh and old leucocytes, proliferated connective tissue cells, lymphocytes, red blood corpuscles, epithelial cells, all of which are more or less degenerated, also pieces and shreds of separated and fibrillated connective tissue, bone-sand, etc.

The pyogenic bacteria are the chief causes of suppuration. It can be produced experimentally by the injections of chemical agents (turpentine, mercury, petroleum, etc.), but such inflammation is never progressive, always remaining localized.

It is difficult to determine why fibrin is frequently not found in pus; possibly the bacterial toxins prevent its precipitation. A fibrinopurulent exudate is frequently seen in tuberculosis, also in purulent inflammations of the serous cavities and joints. A thin yellowish, cloudy exudate, rich in cells, is called a seropurulent exudate. The character of the pus differs in different infections. Staphylococcic pus is creamy and yellowish; streptococcic and pneumococcic pus is thinner and yellowish green; the pus produced by the bacillus pyocyaneus colors the skin and bandages bluish green; tuberculous pus is thin and flocculent; typhoid pus is brownish, thin, and contains pieces of necrotic tissue.

4. *Serofibrinous* and *purulent exudates* which contain considerable quantities of blood are called hæmorrhagic exudates (tuberculous pleuritis, pericarditis, general hæmorrhagic infection, severe inflammations of mucous membranes).

5. In the *ichorous* or *putrefactive* inflammation, due to putrefactive bacteria, destruction of the tissues and putrefaction (gangrene) predominate. The exudate in the beginning is serous and contains few cells, later it is bloody and mixed with detritus and gas bubbles, and therefore becomes discolored and stinking (foul-sanies).

Results of Inflammation.—In any of these forms of inflammation the necrosis of the tissues may be very extensive. Necrosis is extensive, for example, in local anthrax infections, in severe streptococcic phlegmons with a serous or seropurulent exudate, in inflammations of the mucous membranes due to the diphtheria or typhoid bacillus or pyogenic cocci with fibrinous exudate, in suppurative phlegmons and osteomyelitis due to pyogenic bacteria.

An inflammation may pursue an acute or chronic course. As a rule, the difference in clinical picture depends upon the cause of the inflammation. A concentrated caustic produces a more violent inflammation than one that is diluted, but the latter, if applied frequently, maintains a certain degree of inflammation for some time. The same is true of bacterial inflammations, which may be rightly compared to the chemi-

cal inflammations, because the toxins play such an important rôle. Foreign bodies and necrotic tissue—for example, a sequestrum following a suppurative osteomyelitis—irritate continually the surrounding tissues, and for this reason a chronic inflammation persists. While acute inflammation is associated with hyperæmia, exudation, and often with extensive necrosis, these processes are not marked in chronic inflammations, which develop slowly, and often not to any great extent. The proliferative processes are most pronounced in the chronic inflammations, and as a result of the proliferation of the fibrous tissues, periosteum, bone, and adenoid tissue, growths slowly develop which may attain considerable size (e. g., elephantiasis of the skin, callosities, connective tissue thickenings of serous membranes, hyperostoses, chronic tonsillar and lymphadenoid hyperplasia).

In a number of bacterial infections the proliferation of connective tissue about the inflammatory focus is so great that nodular granulation growths, the so-called granulation tumors or granulomas develop, as, for example, in tuberculosis, syphilis, leprosy, chronic glanders, actinomycosis, and rhinoscleroma. These granulation growths contain the specific cause of the inflammation, which invades the normal tissue and gives to the disease in question its characteristic features. If in these chronic inflammations an exudate is formed (e. g., in tuberculosis, seropurulent or serofibrinous effusions in joints and body cavities, hydrops of the joint in syphilis) they pursue a chronic course, and there are only slight symptoms of inflammation.

Chronic interstitial inflammation of muscles and viscera is followed by an atrophy of the parenchyma due to the contraction of the newly formed connective tissues (muscle scar, contracted kidney, cirrhosis of the liver).

LITERATURE.—*Buchner.* Natürliche Schutzeinrichtungen des Organismus und deren Beeinflussung zum Zweck der Abwehr von Infektionsprozessen. Münch. med. Wochenschr., 1899, Nos. 39 and 40.—*Cohnheim.* Vorlesungen über allgem. Pathologie. Leipzig, 1882.—*Graser.* Die erste Verklebung der serösen Häute. Chirurg.-Kongr. Verhandl., 1895, II, p. 625.—*Heidenhain.* Zur Lehre von der Lymphbildung. Archiv f. d. gesammte Physiologie, Bd. 49, 1891, p. 209.—*Herxheimer.* Fibrinöse Entzündungen. Virchows Archiv, Bd. 162, 1900, p. 443.—*Landerer.* Zur Lehre von der Entzündung. v. Volkmanns Samml. klin. Vorträge, 1885, No. 259.—*Leber.* Die Entstehung d. Entzündung. Leipzig, 1891.—*Lubarsch.* Entzündung. Ergebnisse der allgem. Path. v. Lubarsch u. Ostertag, 3. Jahrg., 1896, p. 611.—*Muscatello.* Zur Frage der Entzündung u. Verwachsung seröser Häute. Münch. med. Wochenschr., 1900, p. 688.—*Friedr. Müller.* Ueber d. Bedeutung d. Selbstverdauung bei einigen krankhaften Zuständen. 20. Kongress d. inn. Med., 1902.—*Pappenheim.* Zur Plasmazellenfrage. Virchows Arch., Bd. 169, 1902, p. 372.—*v. Recklinghausen.* Allgem. Path. d. Kreislaufs u. d. Ernährung, Stuttgart, 1883.—*Ribbert.* Lehrbuch d. allgem. Path. Leipzig, 1905;—Die Bedeutung der Entzündung. Bonn, 1905.—*Ritter.* Die Entstehung der entzündlichen Hyperämie. Mitteil. a. d. Grenzgebieten, Bd. 12, 1903, and Bd. 15, 1905.

—*Schlesinger.* Ueber Plasmazellen und Lymphozyten. Virchows Arch., Bd. 169, 1902, p. 428.—*Virchow.* Die Rolle der Gefässe und des Parenchyms in der Entzündung. Virchows Arch., Bd. 149, 1897, p. 381.—*Ziegler.* Entzündung. Eulenburgs Realenzyklopädie, Bd. 7, 1895.

CHAPTER III

THE GENERAL REACTION

THE general reaction following a local infection begins with the diffusion of the bacteria and their toxins in the body, the first symptoms being usually the direct result of absorption. Bacteria pass so rapidly from a recent infected wound into the blood, and from this into the viscera that their direct entrance into the injured capillaries must be considered as probable. The absorption by the lymphatics, and the deposition of bacteria in the lymph nodes, also play a great rôle. Pathogenic are absorbed as rapidly as the saprophytic bacteria (Schimmelbusch, Noetzel).

Rapidity of Absorption from Different Kinds of Wounds.—Only fresh wounds, the incised more than contused or lacerated wounds, have this property of immediate absorption. An old wound, over which the plasma has formed a protective covering, gangrenous and uninjured granulating wounds do not absorb. A rapid absorption of bacteria and their toxins occurs when uninjured mucous membranes are invaded by highly virulent bacteria (Lexer, Bail, *vide* Infection Atria of Pyogenic Infections). The different tissues of the body behave very differently as regards the absorption of bacteria from recent infected wounds (e. g., infection of operation-wound). The peritoneum absorbs most rapidly, and therefore is more resistant to mild infections than the subcutaneous tissues or synovial membranes, while general infections follow rapidly virulent infections of the peritoneum. In the later course of any infection the invasion of the lymphatic vessels and blood vessels by bacteria plays an important part in the diffusion of bacteria and their toxins and the development of a general infection.

Experiments have demonstrated that absorption from fresh wounds begins before the local reaction, and that it continues until the inflammatory focus is encapsulated, until the bacteria are removed or escape is provided for them by operative procedures, or they are deposited in the lymph glands, blood and organs, where they come in contact with and are destroyed by the bactericidal substances. General infection is prevented in mild infections or infections of average severity only, and in these cases the local reaction protects the infection atria. According to the

later investigation it is doubtful whether there is a physiological secretion of bacteria by the kidneys, without any disease of the same, and by the sweat glands (*vide* Lenhartz, Wrede).

Relation of Clinical Symptoms to Virulence of Bacteria and Lowered Resistance of Organism.—It depends upon the virulence of the bacteria whether the clinical symptoms of general infection begin early or late, or whether they develop at all. While the absorption of highly virulent bacteria such as, for example, those derived from an inflammatory process in another patient, give rise to immediate symptoms, those coming from the outer world must become adapted to the tissues, hold their own against the bactericidal properties of the latter and develop, before their absorption and entrance into blood vessels give rise to general symptoms. The bacteria are not able to develop in many cases when introduced into wounds, and when they do a period of incubation is necessary (*vide* p. 146).

If the absorbed bacteria are too virulent or are present in too large numbers, or the organism is weakened and its resistance therefore reduced, they will not be destroyed, and as a result they will either be deposited in the tissues (bacterial metastasis) or develop in the blood and flood the organism (blood infection). While all these processes may occur with one form of bacteria—e. g., pyogenic cocci—the process may be entirely different with another form. For example, in glanders metastasic foci are the rule; in anthrax there is a general blood infection; in tetanus and diphtheria bacteria are but rarely found in the blood and tissues, the infection being toxic, while in miliary tuberculosis, which may follow directly the rupture of a tuberculous focus into a large vessel (e. g., a branch of the pulmonary vein), great numbers of metastatic foci develop in the different viscera and tissues.

The most virulent general bacterial infections characterized by development of bacteria in the blood occur only when all the natural protective powers of the body are greatly reduced or exhausted.

Bactericidal Substances in Tissue Fluids.—The bactericidal substances which give to the organism natural protective powers are normally present in the lymph, blood, and tissue fluids. They are produced by the activity of certain cells, especially the leucocytes. The greater part of the natural resistance depends upon these bodies. Blood serum taken from a healthy body has the property of dissolving or agglutinating a number of different varieties of bacteria, and the blood corpuscles of another species. The substances which do this are called from their action bacteriolysins, hæmolysins, bacterioagglutinins, hæmagglutinins, and, according to Behring, this action is due to a single albuminous-like body which is contained in the serum, the *alexin* (from ἀλέξειν, meaning to protect). The alexin is so unstable that it is rapidly de-

stroyed after being taken from the body, and becomes inert immediately when heated to 55° C. According to Ehrlich and Morgenroth this bactericidal and agglutinating property of serum does not depend upon a single substance, the alexin, but upon the combined action of different bodies. Experiments have demonstrated that the action of the serum depends upon two bodies, one of which, the complement (corresponding to the alexin), is thermolabile and quickly destroyed when the serum is heated to 55° C.; while the other, the intermediary body which forms the bond of union between the bacteria or red blood corpuscles of another species and makes possible the digestive or fermentlike action of the complement, resists heating.

These two bodies are multiple, and different intermediary bodies and complements are present in the serum which are specific for different bacteria and cells, and even for different species (typhoid, cholera bacilli, and blood corpuscles of different species). For this reason a serum which has agglutinated typhoid bacilli can still agglutinate cholera bacilli, but is no longer able to act upon typhoid bacilli.

A serum the complement of which has been destroyed by heat is inactive. It, however, may still contain the intermediary body, and becomes active again as soon as normal serum containing the complement is added to it. The inactive serum is then reactivated.

Normally there are only small amounts of the intermediary bodies in the serum. They are, however, formed during an infection, and when the infection subsides are present in large amounts within a short time. This increase does not affect all the intermediary bodies, however, but only those which are active against the bacteria producing the infection. These are then called immune bodies, for they give to the serum its most important bactericidal and agglutinating properties. If an animal has been immunized against typhoid, its serum will dissolve typhoid bacilli, but only these, as the immune body is specific for the typhoid bacillus. In the immunization the complement is not increased. (Concerning the production of the immune body, see Ehrlich's theory.)

The action of the normally present or newly formed protective substances is successful only when the bacteria are not too virulent and do not multiply too rapidly. In virulent infections with rapid course the bactericidal bodies do not suffice, and are not formed in large enough quantities.

Decrease of Protective Substances.—The decrease of the protective bodies, especially of the complement, predisposes to infection—e. g., if the complement has been exhausted by infection or sufficient quantities are not produced because of disease of the internal organs (Ehrlich and Morgenroth). Von Dungern has made the very significant observation that the complement becomes united with dead tissue, and

explains in this way the increased local predisposition to infection after injury.

Local Increase in Protective Substances Artificially Induced.—A local increase of these protective bodies may be produced artificially. The organism responds to any irritation by hyperæmia, and then increased amounts of the bactericidal bodies of the blood are carried to the injured area, and when the blood stream slows (*vide* Inflammation) these pass out into the tissues. A local accumulation of intermediary bodies and complement may be produced by inducing an artificial hyperæmia or increasing a preëxistent hyperæmia (Wassermann). The favorable influence which those agents producing a mild hyperæmia (tincture of iodine, alcohol, compresses and poultices) exercise upon mild and chronic inflammation, depends mostly upon this increase in intermediary bodies and complement. These same agents do harm in acute inflammations, as the hyperæmia, and consequently the exudate, is increased, and the formation of pus and the digestion of the tissues is hastened or grave circulatory disturbances followed by extensive necrosis are induced.

A large accumulation of bactericidal substances accompanies the passive (venous) hyperæmia, which is recommended by Bier as a therapeutic measure in the treatment of infections, especially for tuberculosis of the extremities, gonorrheal arthritis, and acute inflammations of all kinds. Its use, however, may be compared to that of a two-edged sword, for the good which follows the exudation of blood plasma and the emigration of leucocytes may be counterbalanced by the nutritional disturbances which follow too long and severe compression and the slower absorption of toxins from the inflammatory focus which injure the tissues. Clinical experience, and the animal experiments performed by Nötzel, have demonstrated that passive hyperæmia may under certain conditions prove injurious. Even if properly controlled, passive hyperæmia may do harm, for after the constrictor is removed there is an increased absorption of the pyogenic endotoxins which have been liberated by bacteriolysis and an increase of proteolytic ferments, which are derived from the leucocytes. Passive hyperæmia is especially injurious in virulent and acute infections which have lasted some time (Lexer).

A vigorous, active hyperæmia such as that produced by Bier's hot-air apparatus hastens absorption, and for this reason acts favorably in many cases of chronic articular rheumatism, chronic œdema, etc. (Bier).

The Source of Bactericidal Substances.—The source of the bactericidal substances found in the blood serum has not been accurately determined. According to Buchner and Metschnikoff, the complement or alexin is derived from the leucocytes; Buchner regarding it as a secretion, Metschnikoff as a degeneration product. A number of experiments,

such as those of A. Wassermann, who has produced an anticomplement by immunizing animals with leucocytes, and moreover the increase in the bactericidal properties of an exudate by artificially increasing the number of leucocytes, indicate that the latter is one, but not the only source of the complement (A. Wassermann). The experiments of Ehrlich and Morgenroth have shown that other cells—e. g., liver cells—may produce complement, for if the liver is artificially excluded from the circulation the amount of complement is reduced.

Relation between Leucocytes and Complement.—The relation between the leucocytes and the complement explains the value of two phenomena occurring in infections, the accumulation of leucocytes at the point of invasion (*vide* Inflammation) and the increase of leucocytes in the blood (active leucocytosis), which occurs in almost all febrile infectious diseases (with the exception of typhoid, measles, and malaria), and also when infectious substances from suppurative or putrefactive foci are absorbed. These phenomena may be produced experimentally if irritating substances which attract the motile leucocytes are injected into some part of the body or into the blood. This process of positive chemotaxis (or chemotropism) may be induced by a number of chemical agents and bacterial poisons, particularly by the bacterial proteins, albuminous substances produced in the degeneration of the bacterial protoplasm (Buchner, Römer). These poisonous substances derived from bacteria attract the leucocytes and produce a local accumulation in the inflammatory focus, and after their absorption an increase in the blood of those cells which give rise to the bactericidal substances. The absence of leucocytosis in suppurative processes or general infections indicates either that the organism can successfully combat without effort the bacteria and their toxins or that the organism is too weak to continue the struggle. The same relations hold true at the point of infection (*vide* Pyogenic Infections).

Leucocytosis.—In leucocytosis there is an increase of white corpuscles to over 10,000 in 1 c.mm. of blood, without a decrease in the number of red corpuscles. That variety of leucocyte which is normally present in greatest numbers (sixty-five to seventy per cent) and which in inflammation emigrates in greatest number from the blood vessels, the polymorphonuclear neutrophile leucocytes, is increased. They differ from the other leucocytes in that their protoplasm stains only with the neutral, not with the acid or basic dyes; their nucleus is irregularly lobulated, of horseshoe or clover leaf shape, and stains deeply with basic dyes. According to Ehrlich, they are formed mostly in the bone marrow, but also to some extent in the lymphatic system (E. Grawitz).

Diagnostic Significance of Leucocytosis.—Leucocytosis has been used for diagnostic purposes (Curschmann) to recognize deep-seated suppura-

tion, particularly suppuration associated with appendicitis. But a moderate leucocytosis may accompany the formation of any inflammatory exudate (e. g., pneumonia), and may be absent in large intraperitoneal abscesses, apparently because encapsulated. Leucocytosis is therefore of much less diagnostic significance than the clinical symptoms. [Leucocytosis is of value in diagnosis when it is considered with the clinical symptoms.]

Leucocytes are also able by their amœboid movements to surround organic particles (dust, pigment, detritus, bacteria) and to ingest them. According to Metschnikoff's idea of phagocytosis, the leucocytes wage active warfare against the bacteria, ingest living bacteria and destroy them. Leucocytes are therefore called eating cells or phagocytes. According to Weigert, Buchner and others, virulent bacteria are not ingested by leucocytes, but only those which have been previously injured or killed by the bactericidal substances in the blood serum. Certain micro-organisms (gonococci, leprosy and tubercle bacilli), however, multiply within the leucocytes, and must, therefore, have been viable when ingested.

Infected tissues absorb not only bacteria, but their poisons (toxins and endotoxins) as well.

Ehrlich's Side-chain Theory, Action of Immune Sera, etc.—It is only possible to demonstrate experimentally the presence of toxins in the blood shortly before or after death, and when they are produced in large quantities, as in tetanus and diphtheria. As a rule, the absorbed toxins do not remain in the blood unless produced in large amounts in severe cases of the diseases above mentioned. The toxins have a specific relation to certain cells by which they are attracted and bound, and upon which they act. Tetanus toxin, for example, which experimentally is bound by the substance of the central nervous system of susceptible animals, is drawn from the blood by the nerve cells and acts upon them. In unsusceptible animals the toxins circulate in the blood, producing no symptoms, as they are not attracted and bound by the cells.

The union of the toxin with the cell is explained by Ehrlich in the following way: The toxin molecule possesses a haptophore and a toxophore group. By means of the former the toxin becomes attached to the cell, and then the toxic properties residing in the latter become active. In Ehrlich's theory the activities of the living cell reside in a " Leistung-kern " (action center or nucleus) and in different " Seitenfunctionen " or side-chains (receptors), which bind and assimilate the food substances. The toxin is bound as follows: Its haptophore group becomes attached to definite side-chains or receptors of certain cells, into which the haptophore group of the toxin fits like a key in a keyhole, using the comparison made by E. Fischer. The susceptible cells which

bind the tetanus toxin, for example, are found in the central nervous system, and the toxin becomes united with these cells and acts upon them. The location of the cell groups which bind the toxins in other infections is unknown as yet.

Ehrlich's side-chain theory also explains the complicated processes which provide for the production of antitoxins and the immunization of the individual.

If an animal has been rendered immune against tetanus by the administration of gradually increasing doses of tetanus toxin, so that the fatal dose of the toxin can be borne without harm, the blood serum of the immunized animal will protect another animal from the ordinarily fatal action of tetanus toxins or infections. The serum now contains an antitoxin for tetanus which binds the toxin and renders it harmless. This was first demonstrated for tetanus in 1890 by Behring and Kitasato, and has become generally known in experimental work as "Behring's law." The blood serum of an individual rendered immune spontaneously (by disease) or artificially (by inoculation with living cultures or with toxins) against a certain disease, when injected transfers the immunity against the disease in question to a susceptible individual. Active immunization, produced by the injection of increasing doses of toxic substances against which antibodies are formed, is differentiated from passive immunization, which is produced by the injection of the blood serum of an animal already immunized. Active immunization resembles acquired immunity, which occurs when a patient recovers from a disease. As in tetanus, so in diphtheria, immunity depends upon the production of antibodies, which act upon the toxins. In other infections the antibodies do not act upon the toxins, but upon the bacteria themselves. Animals may be immunized against poisons other than those produced by bacteria. They may be immunized against toxic albuminous-like substances derived from plants, such as ricin and abrin (Ehrlich), against snake venom (Calmette), not, however, against other toxic substances (alkaloids).

According to the side-chain theory, in the immunizing process (as in natural infections) the toxins which are injected become united with the susceptible cells, in tetanus, for example, with the cells of the central nervous system, for the side-chains or receptors of the cells become firmly attached to the haptophore group of the toxin. The toxin does not produce fatal results, for in the beginning of artificial immunity only small attenuated nonlethal doses, which are gradually increased, are injected. The side-chains or receptors which are bound by toxin no longer functionate, and are no longer of any use to the cell. According to Ehrlich, when the side-chains are bound new side-chains are formed, not simply to replace those already destroyed, but in excess, this explanation being

based upon the hypothesis advanced by Weigert that in regeneration after injury tissues tend to reproduce not only to the extent of repairing the injury, but in excess. Only those side-chains which are required to replace those destroyed remain attached to the cell, the remainder are thrown off into the blood. The blood, as well as the susceptible cells, now contains side-chains or receptors which are able to bind the toxin. While the side-chains attached to the cells render possible the action of the toxins upon the cell, the free side-chains circulating in the blood render the toxins harmless as they become united with the latter, which can then no longer reach the susceptible cells and destroy them. The excessive side-chains or receptors which are cast off into the blood therefore form the antitoxin and the nucleus of every immune serum.

Antitoxic Serum.—Antitoxic serum (e. g., in tetanus, diphtheria) neutralizes only the toxin. The bacteria remain viable, and are gradually destroyed by the bacteriolytic powers of the organism, perhaps also by the saprophytes.

Besides the infections mentioned above in which the antibodies obtained by immunization are antitoxic, there is a second group of infectious diseases in which the blood serum of a patient who has recovered from the disease or of an animal which has been immunized contains specific bactericidal antibodies, which act upon the bacteria themselves without neutralizing their toxins (cholera, typhoid, plague). There is the following difference between these two antibodies when the treatment of infections is considered: Antitoxic sera may be expected to cure the disease, while bactericidal sera have only an immunizing action, and for that reason are only of value in prophylaxis. Bactericidal sera may even do harm if the disease has already developed, for large amounts of endotoxins are set free suddenly by the bacteriolysis which they induce.

Bactericidal Serum.—The bactericidal immune serum (e. g., in typhoid, cholera infection) contains a specific immune body. This is the specific intermediary body already present in the serum, but which has been increased by immunization (*vide* p. 156). The immune body becomes attached on one side to the bacteria, and on the other to the complement already present in the normal serum, and renders possible the digestive action of the latter upon the bacteria. A small amount of such immune serum injected into a normal animal suffices to protect it, for the bacteria which ordinarily would produce fatal results are dissolved in the tissues. Immune sera taken from the body lose their power after a few days, for the labile complement is rapidly destroyed outside the body. As soon, however, as fresh serum from a normal animal is added, the power returns. New complement has been added and the immune body is still in the serum (Metschnikoff).

The production of the specific immune body is explained by Ehrlich's

theory in the same way as the production of antitoxin. The toxic substances produced partly by bacterial metabolism and partly by bacteriolysis find in the cells specific side-chains. The bound side-chains are replaced in excess, and the excessive side-chains are thrown off into the blood. These side-chains, which are the immune bodies, become bound to the bacteria and render possible the digestive action of the complement. The source of the immune body for all infections is not known. According to Pfeiffer, Marx, and A. Wassermann, the medulla of bone, the spleen, and lymph glands may be regarded as the origin of the immune body in cholera and typhoid fever; according to A. Wassermann, the medulla of bone in pneumonia. In the medulla of bone are cells which are able to become bound with bacteria. (Cf. Pyogenic Diseases of Bone.)

Besides these specific immune bodies, by the aid of which the complement dissolves bacteria, many immune sera or the blood of sick or convalescent patients contain substances which agglutinate bacteria. These substances are called specific agglutinins. In typhoid fever they are found in the blood early (Widal); they are also found in tuberculosis (Koch) and in staphylococcic and streptococcic immune sera, etc.

Experimentally, not only specific immune bodies for bacteria, but also for all varieties of cells of another species may be produced, for red and white blood corpuscles, ciliated epithelium, and spermatozoa. These immune sera will dissolve the cells of the species in question and partly agglutinate them. As a result of this, the action of hæmolytic (dissolving red corpuscles) and leucotoxic (dissolving white corpuscles) sera is fatal. (Cf. A. Wassermann.)

The endotoxins (bacterial protoplasmic poisons), the most important poisonous substances of pyogenic bacteria, are absorbed in addition to the toxins. The organism is able to neutralize certain quantities of endotoxins, but it is unknown in what way this is accomplished.

Action of Toxin and Endotoxin.—The action of these toxic substances (toxin and endotoxin) produced by bacteria is harmful, excepting the reactive processes which produce protective substances. The results of the action of these substances differ, depending upon the tissues involved, the variety of toxin, and the amount produced. The results are most striking in tetanus, in which the clinical picture is produced by the direct action of the toxins upon the cells of the central nervous system or in the toxic neuritis, resulting in paralysis, which occurs in diphtheria. Cerebral symptoms, which must be attributed to the action of toxins, are pronounced in many acute and severe infectious diseases and wound infections. Sometimes these are confused with the symptoms of the fever and are regarded as constant features of it. Other changes in the nervous system, such as myelitis, neuritis, neuralgia

and neuroses; gastric disturbances; and albuminuria are in all probability due to the action of toxins.

Hæmolysis.—Many toxic substances, such as those produced by the tetanus bacilli, strepto- and staphylococci, dissolve red corpuscles, and increased amounts of urobilin are therefore found in the urine in these infections. As a result of this and of the weakening of the organism, anæmia occurs in chronic suppuration (osteomyelitis, general infection) and in chronic diseases (tuberculosis, actinomycosis, syphilis, etc.). Parenchymatous and amyloid degeneration of organs (heart, liver, and kidneys) are also due partly to the action of toxins.

LITERATURE.—*Aschoff.* Ehrlichs Seitenkettentheorie und ihre Anwendung auf die künstlichen Immunisierungsprozesse. Zeitschr. für allgem. Physiol., Bd. 1, 1902. —*Behring.* Allgem. Therapie der Infektionskrankheiten. Urban u. Schwarzenberg, 1899.—*Buchner.* Ueber Immunität. Deren natürliches Vorkommen. Münch. med. Wochenschr., 1891, p. 551.—*Curschmann.* Zur diagnost. Beurteilung der vom Blinddarm, etc., ausgehenden entzündl. Prozesse. Münch. med. Wochenschr., 1901, p. 1907.—*Friedberger.* Die bakterizid. Sera. In Kolle-Wassermanns Handb. der path. Mikroorg., Bd. 4, p. 452.—*Friedrich.* Die aseptische Versorgung frischer Wunden. Chir.-Kongr. Verhandl., 1898, II, p. 46.—*Küttner.* Diagnost. Blutuntersuchungen bei chirurg. Eiterungen. Chir.-Kongr. Verhandl., 1902, I, p. 126.—*Lexer.* Zur Behandlung akuter Entzündungen mittelst Stauungshyperämie. Münch. med. Wochenschr., 1906, p. 633.—*Metschnikoff.* Die Lehre von den Phagozyten und deren experimentelle Grundlagen. In Kolle-Wassermanns Handb. der path. Mikroorg., Bd. 4, p. 332.—*Moxter.* Die Beziehungen der Leukozyten zu den bakterienauflösenden Substanzen tierischer Säfte. Deutsche med. Wochenschr., 1899, p. 687.—*Nötzel.* Ueber d. Bakterienresorption frischer Wunden. Arch. f. klin. Chir., Bd. 60, 1900, p. 25.—*Oppenheimer.* Die Bakteriengifte. In Kolle-Wassermanns Handb. der path. Mikroorg., Bd. 1, p. 344.—*Paltauf.* Die Agglutination. Ibid., Bd. 4, p. 645.—*Römer.* Die Ehrlichsche Seitenkettentheorie und ihre Bedeutung für die med. Wissenschaften. Wien, 1904.—*Sachs.* Die Hämolysine und ihre Bedeutung für die Immunitätslehre. Wiesbaden, 1902.—*Schimmelbusch* und *Ricker.* Ueber Bakterienresorption frischer Wunden. Fortschr. der Med., Bd. 13, 1895.—*A. Wassermann.* Weitere Mitteil. über Seitenkettenimmunität. Berl. klin. Wochenschr., 1898, p. 209;—Wesen der Infektion. In Kolle-Wassermanns Handb. der path. Mikroorg., Bd. 1, p. 223;—Antitoxische Sera. Ibid., Bd. 4, p. 452;—Hämolysine, Cytotoxine und Präzipitine. v. Volkmanns Samml. klin. Vortr. Natur Forscher, No. 331.—*Wrede.* Die Ausscheidung von Bakterien durch den Schweiss. Chir.-Kongr. Verhandl., 1906.

CHAPTER IV

FEVER

ALL the reactive processes which follow the absorption of bacteria and their toxins have for their object the control of the infection. The general reaction is expressed clinically by fever.

Symptoms of Fever.—The chief symptom of fever is an elevation of body temperature. The normal body temperature, when taken by the mouth is 98.6° F., in the rectum 99.6° F. In mild fevers the temperature reaches 101° F. and in severer ones 104° F., or even 1° to 1.5° higher. Disturbances of digestion (anorexia, vomiting); of the circulation (rapid and soft pulse); and of the respiration; and nervous symptoms (irritability, headache, disturbance of consciousness, delirium) often accompany fever. These vary with the temperature, and are caused less by fever than by the bacteria which produce it.

The elevation of temperature results from a disturbance of the equilibrium between heat production and heat loss. In the healthy individual as much heat is lost by radiation and conduction from the skin and by evaporation from the skin and lungs, as is produced, for example, by oxidation processes occurring in the muscles. In fever the production of heat is increased, and the amount lost is not sufficient to maintain the equilibrium. The increased production of heat depends upon increased metabolism and oxidation processes, for in fever the amount of oxygen contained in the expired carbon dioxid (Liebermeister, von Leyden) is greater than that which is inhaled (Regnard, Zuntz), and because of the increased destruction of albumins the nitrogen excreted in the urine is increased. In a healthy individual increased metabolism (muscular effort) does not produce an elevation of temperature, because the excessive heat is lost by radiation, conduction, or evaporation.

In fever the processes which effect this are disturbed. The loss of heat does not keep pace with its production. There is interference with the loss of heat, so that in the beginning this may be less than normal, and thus there is produced a heat congestion (Traube).

According to Krehl, it is most probable that the substances which produce fever incite abnormal decomposition processes. Thus there is produced an increase in the decomposition and oxidation of albumins which interferes with the loss of heat. It may be that the stimulus which excites heat loss is deficient, or that the mechanism (blood vessels, sweat glands, and breath) which effects it functionates poorly, or finally that the regulating center in the brain is at fault.

A sudden rise of temperature (stadium incrementi) is often accompanied by a feeling of chilliness or a rigor. The amount of heat lost is diminished, while the production of heat is increased in this stage. The capillaries of the skin are contracted by the action upon the vasomotor centers of the fever-producing substances. Reflex muscular twitchings may occur and give rise to the clinical picture of a chill.

In the climax (fastigium) which follows after one to two hours, the skin is white, dry, and somewhat injected. The amount of heat lost is

relatively increased, but not enough is lost to bring about a return to the normal condition.

The fall of the fever (stadium decrementi) is frequently accompanied by marked sweating and sometimes by symptoms of collapse. Heat is not formed in such large amounts, while the loss of heat is effected in a number of different ways. If, during an infection, the organism becomes weakened and its resistance decreases, the temperature falls; for example, if, as a result of cardiac weakness, sufficient heat is not produced. Collapse, which frequently proves fatal, may then develop.

Surgical Significance of Fever.—1. Particular fever curves give to many infections a characteristic impress which is of great diagnostic importance. 2. An elevation of temperature indicates some irregularity in wound repair, the beginning of invasion by micro-organisms or the accumulation of inflammatory products.

Causes of Fever.—The fact that the temperature rises with the development of an abscess or with the beginning of suppuration or putrefaction in a wound, and that it subsides when the pus is permitted to escape or the inflammation subsides, is proof that the substances producing the fever result from the inflammation or are contained in the wound secretion. At a time when little was known about bacteria, and nothing was known about their action, experiments were made with putrefactive wound secretions and the decomposition products of animals and plants (Billroth, Weber, von Bergmann) to determine the cause of fever following wound infections.

Not only the decomposition products from suppurative and putrefactive foci, but a number of other substances which produce an increased decomposition of albumins (Krehl) cause fever. All ferments (fibrin ferment, according to von Bergmann and Angerer), poisonous albuminous substances, the decomposition products of cells (hæmoglobin), foreign constituents of the blood (cells and serum) of man and animals, and especially the toxins of pathogenic bacteria cause fever.

Relation of Fever to Absorption of Toxins.—*Decline Due to Protective Substances.*—It has been demonstrated by experiments upon animals and man and by clinical experience that the diffusion of the toxins of pathogenic bacteria in the body produces fever, and that the decline of the fever follows the development of protective substances in the blood. The fever in diphtheria subsides as soon as antitoxin is injected, while after the crisis in pneumonia protective substances are present in the blood. Depending upon whether the antibodies are developed quickly or slowly, the fall of temperature is sudden (critical) or gradual (lytic).

In continuous fever, in which the difference between the maximum and minimum rises of temperature taken morning and evening is at most one degree, the poisonous products of the bacteria predominate over anti-

bodies. In intermittent fever, in which there are intervals of days without fever, antibodies are formed intermittently, are then exhausted, and allow the newly formed toxins to act. The same holds true for remittent fever in which the fever falls in the morning.

The different forms of fever curves in the different infections depend upon the bacteria producing the infection, the toxins which are formed in the tissues, and the products of decomposition (ferments in the exudate, dead tissues which are dissolved) resulting from the inflammation. For this reason many diseases have typical fever curves, in which a high fever persists for a certain time and then falls; in pneumonia and erysipelas, for example, in about one week the organism overcomes the infection by oxidizing the products of decomposition and by producing protective substances.

The examination of the cells (medulla of bone, spleen, and lymph glands) of an organism which has formed bactericidal bodies in a number of infections will reveal increased cellular activity, as indicated by relatively numerous karyokinetic figures (Pfeiffer, Marx, A. Wassermann, Freymuth), showing that an effort has been made in this way to overcome the infection.

Fall of Fever after Incision of Abscess, Amputations, etc.—The fall of temperature after the incision of a phlegmon or abscess or after the amputation of a suppurating extremity indicates that the toxins and products of decomposition are no longer being absorbed. If the fever rises again, it indicates that new tissue is being invaded or that the discharge of pus is prevented. If the infection is not controlled by incision or amputation the fever continues until death, the overwhelming infection producing continuously new toxins before sufficient protective substances are formed to prevent their fatal action (*vide* p. 156).

Low Fever in Fatal Infections.—Fever may be absent or slight in fatal infections. Because of the virulence of the bacteria or the weakness of the organism (old people) there is no general reaction. In animal experiments there is often no fever after the injection of large fatal doses of toxins, but subnormal temperature and collapse. Fever, therefore, not only indicates the beginning and extension of severe infection, but indicates during its entire course the activity of those processes which combat infection.

Some importance has justly been attributed to the harmful action which fever exerts upon bacteria. Many bacteria, particularly the gonococci, are killed when exposed to high temperature. The conditions in the culture tube, however, are not comparable to those in the living body, for in the former the bacteria are exposed to the desiccating action of the air, and for this reason this supposed action of fever is doubtful (A. Wassermann).

Action of Antipyretics.—The more one considers the value of the febrile reaction, which was claimed by Hippocrates, but which has been doubted more recently, the less one is inclined to prescribe chemical agents (antipyretics) to control the fever. One fears that they will interfere with the production of protective substances, although Schütze has demonstrated at least for typhoid fever that antibodies are formed even when the temperature is reduced by antipyrin. The surgeon should not, however, use antipyretics to reduce the fever, for next to the general appearance of the patient, it is the most important index of the condition of the wound or inflammatory focus.

Aseptic Fever.—A non-infectious or aseptic fever (Genzmer and von Volkmann) is distinguished from fever resulting from the absorption of infectious substances, especially bacterial toxins. This fever occurs after subcutaneous injuries, especially after fractures; with vascular sarcomas; when there is extravasation of large quantities of blood into the tissues; and after injuries of certain parts of the brain. It does not occur constantly, however, and differs clinically from the infectious fever in the absence of a chill and general febrile symptoms. The pulse is good and only slightly accelerated, and there is but little elevation of temperature (100° F., rarely higher).

This fever is caused by the formation and absorption of pyrogenic substances from blood exudates and dead tissue. Earlier it was suggested by Alex. Schmidt, von Bergmann, and von Angerer that the fever was caused by the fibrin ferment. According to the later investigations of Schnitzler and Ewald, it is produced by the nucleins, albuminoses, and allied substances.

The elevation of temperature after injury of the brain is caused by a disturbance or irritation of certain parts of this organ (e. g., medial part of the corpus striatum). It may be produced experimentally (heat puncture).

A slight elevation of temperature without other symptoms of fever occurs frequently after operation- and open wounds (also after childbirth), although there is no irregularity in wound repair. This has been spoken of as aseptic fever, and the absorption of the decomposition products of injured tissue and of blood exudates has been regarded as the cause (von Volkmann). The demonstration, however, of bacteria in accidental- and operation-wounds which heal without inflammation, likewise the demonstration of slightly virulent pathogenic bacteria upon hands which have been thoroughly sterilized, upon the sterilized skin of the patient, in the air, etc., indicate that bacteria are an important factor in so-called aseptic fever following operation- and accidental-wounds, even when the wound repair pursues an aseptic course. Used in this sense, Volkmann's designation, aseptic fever, may still be retained.

A purely nervous fever, due to an irritation of the central nervous system (heat center), occurs in insanity and psychoses, particularly in paretic dementia and hysteria.

LITERATURE.—*v. Bergmann* und *Angerer*. Das Verhältnis der Fermentintoxikation. Festschr. d. Würzburger Universität, 1882.—*Brunner*. Wundinfektion und Wundbehandlung, I, Frauenfeld, 1898.—*Freymuth*. Exp. Untersuch. über d. Beziehungen leichter Infekt. z. blutbild. Apparat. Deutsche med. Wochenschr., 1903, p. 350.—*Genzmer* und *Volkmann*. Sept. u. asept. Wundfieber. v. Volkmanns Samml. klin. Vortr., No. 121.—*Krehl*. Das Fieber. Path. Physiol., Leipzig, 1904.—*Adolf Schmidt*. Lehrbuch der allgem. Path. und Ther. innerer Krankheiten. Berlin, 1903.—*Schnitzler* und *Ewald*. Beitrag zur Kenntnis des aseptischen Fiebers. Arch. f. klin. Chir., Bd. 53, 1896, p. 530.—*Unverricht*. Ueber das Fieber. v. Volkmanns Samml. klin. Vortr., Natur Forscher, No. 159, 1896.—*A. Wassermann*. Wesen der Infektion. In Kolle-Wassermanns Handb. der path. Mikroorg., Bd. 1, 1903, p. 223.

II. WOUND INFECTIONS PRODUCED BY PYOGENIC AND PUTREFACTIVE BACTERIA AND THEIR RESULTS

A number of different varieties of bacteria are found in wound infections. Those producing suppurative inflammation are grouped as pyogenic bacteria. Closely allied to these, and often associated with them, are the putrefactive bacteria. A second large group includes those bacteria which produce specific diseases (cf. 2, Part III).

The pyogenic bacteria are divided into those which produce suppurative inflammation (pyogenic cocci), and those which rarely produce pus, but more frequently other forms of inflammation or specific diseases (pneumococci, gonococci, bacterium coli commune, bacillus pyocyaneus, typhoid bacilli). No pyogenic bacteria are exclusively pyogenic, on the other hand they are all phlogogenous—that is, they produce inflammation which, with some more frequently than with others, ends in suppuration.

CHAPTER I

THE MOST IMPORTANT PYOGENIC BACTERIA

THE first microscopic demonstration of minute living matter in pus is ascribed to O. Weber (1863) and Rindfleisch (1866). Later von Recklinghausen, Waldeyer, and Klebs (1871), Orth (1872), Birch-Hirschfeld (1873) discovered micrococci in pyæmia, septicæmia, puerperal fever, and suppurative inflammation. In 1874 Billroth described another form, his cocco-bacteria septica.

R. Koch was the first to make an accurate study of the pyogenic bacteria, and his work on wound infections (1878) laid the foundation of modern bacteriology, and the isolation and cultivation of different varieties of bacteria began with the introduction by him of transparent, firm culture media (1881). Ogston (1880–82) described the microscopic appearance of cocci found in pus, and differentiated streptococci from

staphylococci. In 1883 Becker, following Koch's directions, obtained in pure culture a yellow staphylococcus from a case of osteomyelitis, and Fehleisen a streptococcus which caused erysipelas. Pure cultures of a number of different varieties of cocci were obtained by Rosenbach (1884) and Passet (1885) and differentiated from one another, and their etiological significance in the inflammatory processes associated with them recognized.

(a) STAPHYLOCOCCI

(Spherical fission-fungi, occurring usually in grape-shaped colonies; from σταφυλή, meaning grape.)

The staphylococcus pyogenes aureus was first obtained in pure cultures by Becker; later by Rosenbach. The cocci occur mostly in groups, rarely singly or in pairs (Fig. 89).

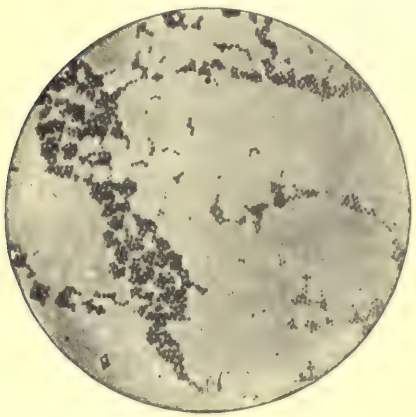


Fig. 89.

They stain readily with basic aniline dyes, and are not destained by Gram's method.

They grow upon ordinary culture media, appearing in gelatin after from thirty-six to forty-eight hours as small white points, in stab cultures as a grayish white deposit. On the third day the gelatin becomes liquefied, and the colonies assume a yellowish color. The entire culture medium is liquefied in three weeks.

Upon agar, after standing for twenty-four hours in the incubator, they appear as round white, later as golden yellow, colonies; the culture medium is not liquefied. They develop similarly upon blood serum and potato. Bouillon is clouded.

The cocci are very resistant, and withstand drying for some days. After remaining in cultures for over a year they are able to develop, and only after being exposed to a temperature of 80° C. for a quarter of an hour are they killed (von Lingelsheim).

The yellow staphylococci are widely distributed.

They may be found alone, or associated with other bacteria in all forms of suppuration or general infection in man, and they are able to develop after being encapsulated in bone for a number of years. They pass into the blood and frequently produce inflammatory foci in young bones; this is partly due to the peculiar way in which they grow, as they form clumps which may occlude the capillaries.

They are found in the skin, the hair, and upon mucous membranes. A wide area of skin surrounding small pustules, furuncles, or a suppurating wound is infected with pyogenic cocci. They may be found upon a surgeon's hands who daily comes in contact with pus or infectious materials. They are found upon the mucous membranes of the upper respiratory passages, without giving rise to inflammation (Miller), occurring especially in the saliva, in the crypts of the tonsil, in the coating of the tongue, and upon the nasal mucous membranes. They can be transferred to the air in small particles of mucus, which are discharged in speaking, clearing the throat, coughing, and sneezing (Fluegge). Apparently they find favorable conditions for growth in the buccal cavity of man. They disappear in a few days when transferred to animals—e. g., to the buccal mucous membrane of the rabbit (Lexer).

Usually, however, staphylococci found upon healthy mucous membranes are attenuated. They may be carried by the food without loss of virulence into the gastrointestinal canal, and in case of perforation or circulatory disturbances (contusion, invagination, or strangulation) cause peritonitis, or, usually associated with other bacteria, inflammation about the rectum (periproctitis). They are frequently found in the conjunctival sac and upon the vaginal mucous membrane.

They are found upon objects surrounding man, and are especially numerous when one is unclean in the treatment of a suppurating focus or inflamed mucous membrane; being then found in the linen, in pocket handkerchiefs, and on all objects with which the patient comes in contact. They have been demonstrated in the dust of the street, in the air of hospitals, but not in the earth or in unconfined air (Passet).

The staphylococcus aureus rarely occurs spontaneously in animals. They have been found in osteoarthritis in geese (Lucet), in osteomyelitis in cattle (Haas) and horses (Fröhner) and in mastitis in cows.

They differ in virulence, both in wound infections and in infection produced in animal experimentation. The virulence of cocci is increased by transmitting them through different animals.

Rabbits and guinea pigs are susceptible; mice and dogs, cows, horses, and goats are less so. Cutaneous inoculations are only successful when the cocci are highly virulent (after cultivation). Subcutaneous injections are followed by the formation of encapsulated abscesses containing thick pus. Usually the inflammatory process is not progressive. Fatal infections are produced only by the injections of cultures into the pleura, peritoneum, or blood vessels. Animals die after intravenous injections in from one to eight days, and suppurating foci are then found in the muscles, viscera, and joints (in young animals foci in bones are found especially frequently, Rodet). If attenuated cultures are used, the

animals run a temperature and are sick for a short time, often developing a chronic suppurative osteomyelitis of one or more bones (Lexer).

It is important to determine the pathogenicity of the different staphylococci (e. g., those upon the skin of sterilized hands, in nonpurulent wound secretion, in the saliva and air). At the present time there is no method which can be relied upon. Animal experiments cannot be relied upon, as the susceptibility of animals and the virulence of the cocci vary. The serodiagnostic test of Kolle and Otto may prove of value in this connection (*vide* below).

The toxins of staphylococci are of two kinds: The toxin (staphylotoxin) demonstrable in culture filtrates and in inflammatory exudates produces local necrosis and suppuration and general toxic symptoms. It is destroyed when heated to 60° C. It is toxic for leucocytes (van de Velde) and dissolves red blood corpuscles, and must therefore contain a leucocidin as well as a hæmolysin (hæmotoxin) (Neisser and Wechsberg). The second is a protoplasmic toxin (endotoxin), which is bound to the bacterial cell, and is found only after the bacteriolysis of large quantities of staphylococci (von Lingelsheim).

Staphylococci also form ferments which digest albumen and gelatin.

Attempts at immunization have given no practical results, although some have succeeded in different ways in immunizing animals, and have obtained from them a serum which was active in normal animals (von Lingelsheim).

Kolle and Otto have used the blood serum of rabbits which had been immunized with large quantities of dead cultures of staphylococci to differentiate the pathogenic from the saprophytic varieties. The serum of an immunized animal has the property, even in dilutions of 1 to 100, to agglutinate in a short time pathogenic bacteria. [Serums obtained by immunization with pathogenic strains have a much higher agglutinating power for these strains than for nonpathogenic varieties, and the converse is also true.—Ricketts' "Infection, Immunity, and Serum Therapy," p. 383.] Nonpathogenic varieties are not agglutinated by serum obtained by immunization with pathogenic varieties. Nevertheless the testing of the agglutinating properties of human serum is not used to determine whether the disease is produced by staphylococci or not, as the serum contains staphyloagglutinin not only in pure staphylococcic infections, but also in infections in which they are secondary to some other variety of bacteria (Beitzke). The formation of antihæmolysins has also been used for diagnostic purposes. According to Neisser and Wechsberg, an antitoxin is developed for the staphylohæmolysin (staphylolysin). This antitoxin, which is called antihæmolysin or antilysin, prevents the action of the lysin. Bruck, Michaelis, and E. Schultze found that in

staphylococcic infections the antilysin content frequently, but not always, exceeded considerably that of the serum of a healthy man.

The **staphylococcus pyogenes albus** was first cultivated by Rosenbach. It differs from the aureus in that its cultures remain white. It is found more frequently than the aureus as the cause of mild inflammations, combined with which it usually causes severe inflammation. It is found almost constantly upon the skin, and frequently produces suppuration about stitch holes and mild wound complications. The staphylococcus albus may be demonstrated constantly in the skin of the hands (*vide* Hand Sterilization, Part I), and frequently upon accessible mucous membranes. Notwithstanding the fact that it occurs much more rarely than the aureus in severe suppurative processes (excepting the double infection with both varieties), it should be remembered that it may produce severe inflammatory changes, even fatal general infections.

In animal experiments it does not differ from the aureus.

The **staphylococcus pyogenes citreus** (Passet), characterized by its color, likewise the **staphylococcus cereus albus** and **flavus,** characterized by the waxy appearance of their white or yellow colonies, are of much less importance. They are only rarely found in human pus. (Hentschel found the staphylococcus citreus in a fatal infection following a furuncle of the lip in the pus, blood, and in the spleen. Jacobitz also found them in a general infection.)

(b) STREPTOCOCCI

(*Streptococci are fission-fungi occurring in chains or pairs.*)

The streptococcus of erysipelas discovered by Fehleisen in 1881 and cultivated by him in 1883, and the streptococcus pyogenes cultivated by Rosenbach in 1884 are not to be regarded as specific for any diseased process, but as closely related. Classifications based upon pathological and clinical symptoms are unreliable, as any streptococcus independent of its origin may, under certain conditions, produce any form of inflammation which is peculiar to streptococcic infections. Mild suppuration or severe general infection may be produced by the streptococci of erysipelas, while other varieties of streptococci, although not derived from an erysipelatous focus, may produce erysipelas (Petruschky). The clinical picture does not depend upon the variety of streptococci, but upon a number of different factors, among which the virulence of the bacteria and the susceptibility of the patient are the most important. The many transitions in the clinical forms of streptococcic infections are explained in this way.

Depending upon the cultural differences upon definite culture media

(blood agar, litmus-metrose agar), Schottmueller and Eug. Fraenkel have differentiated three varieties of streptococci which are pathogenic for man. Of how much bacteriological and clinical importance this distinction is, must be determined by later investigations.

Streptococci are spherical or somewhat flattened, have no movement of their own, and are slightly larger than staphylococci. They always divide in the same direction, and the characteristic slightly tortuous chains of from eight to twenty cocci are formed in this way (Fig. 90). Diplococcic forms are found only in the tissues, inflammatory exudates, in the blood, especially in severe inflammatory processes, and these become transformed in culture media into long chains.

FIG. 90.

They stain with aniline dyes, and according to Gram's method. Streptococci may be most easily cultivated upon agar and blood serum. After twenty-four hours (in the incubator) small, round, somewhat transparent, closely approximated colonies develop, which do not become much larger during later growth. Upon gelatin (at room temperature) they develop much more slowly; small, transparent drops developing after a number of days; a delicate white deposit forming along the tract in stab cultures. Gelatin is not liquefied.

Bouillon is clouded by one variety of streptococci, while a flocculent deposit is formed by another at the bottom of the media.

The streptococci become attenuated or die after a few days upon any kind of culture media, so that they must be transferred daily if their virulence is to be preserved. It is simpler to use Petruschky's method in maintaining virulence, in which two-days-old stab cultures are kept in an ice chest and the same virulence is maintained for months. Streptococci are particularly resistant against drying. They can even be dried upon blotting paper for some time and still retain virulence (Petruschky). The cocci are destroyed in cultures which are heated for an hour at from 70° to 75° C.

Streptococci are as widely distributed outside of the body as staphylococci. However, it is more difficult to demonstrate them on man, in the air of hospital wards and operating rooms, upon objects, skin, and mucous membrane, as they are overgrown by other varieties of bacteria upon culture media. They are found in the same places as the staphylococci.

The frequency of erysipelas and puerperal sepsis in preantiseptic times indicates how easily virulent streptococci may be transferred to wounds or to the vagina and uterus by unsterilized hands and instruments.

Their frequent occurrence in the upper air passages upon healthy as well as upon slightly or severely inflamed mucous membranes shows that they find favorable conditions for growth here. But their presence alone is not sufficient to produce pathological changes; other factors are necessary, such, for example, as an increase of virulence resulting from putrefactive processes or decrease in local resistance from chilling or some injury.

Virulent streptococci are expelled, especially in catarrh, from the buccal and nasal cavity with forcibly expired air (in coughing and sneezing), or they are carried by the saliva and food into the stomach, where they are not always destroyed by the gastric juice. The development of streptococcic peritonitis following perforation of the gastrointestinal tract due to subcutaneous rupture or ulceration, the finding of streptococci in the pus of appendiceal abscesses and in the exudate in the sac of a strangulated hernia demonstrate conclusively that streptococci are carried by the food into the intestines.

Streptococci are found in many different inflammatory processes, and are frequently associated with staphylococci. If the streptococci act alone, a serous exudate into the tissue is in the beginning the most marked feature of the inflammation. Erysipelas is usually a serous inflammation. Frequently in the severe progressive phlegmon due to the streptococci, only a few insignificant purulent foci develop within the inflammatory œdema, necrosis of the connective tissues usually occurring rapidly and becoming extensive. Mild suppurative processes are rarely caused by streptococci. Severe general symptoms develop much more frequently from small cutaneous wounds infected with streptococci than from similar infections with staphylococci.

Streptococci may be found in all the inflammatory processes which are produced by staphylococci. They are found much less frequently, however, in furuncles and osteomyelitis. Mixed infection with the streptococcus, such as occurs in tuberculosis of the lungs, diphtheria, typhoid fever, and putrefactive inflammation, always adds to the gravity of the prognosis.

The virulence of streptococci varies within wide limits in man as well as in susceptible animals. The difference in clinical pictures is largely dependent upon this fact, partly also upon the kind of infection and the individual resistance, which may be reduced by disease (tuberculosis, influenza, diphtheria). The clinical pictures of streptococcic infections differ even in healthy individuals, as the inoculation experiments

of Koch and Petruschky have demonstrated. Streptococci may be attenuated by passage through another species; for example, streptococci from a rabbit may be attenuated for this animal by passing them through a mouse (Knorr). Streptococci derived from man are therefore the most dangerous in human wound infections. This agrees with clinical experience.

White mice and rabbits are the most susceptible of experimental animals. The former succumb in from one to six days of a general infection after subcutaneous or intraperitoneal injections of small amounts of streptococcic cultures. In rabbits the different grades of virulence of streptococci are indicated in the following way: In infection of the wound of the ear with slightly virulent cocci, an erysipelas of moderate severity develops; while if highly virulent cocci are used a general infection without any local changes develops which proves fatal in from twenty-four to forty-eight hours. Different degrees of virulence may be produced in streptococci of different origin by artificially increasing or decreasing their virulence.

When an animal dies some days after the inoculation, streptococci may be cultivated from all the viscera and the blood or demonstrated microscopically. After intravenous injections metastatic foci of suppuration develop in many joints, more rarely in the viscera. Osteomyelitis develops in young animals after the use of attenuated cultures (Lannelongue, Lexer).

Guinea pigs are less susceptible than rabbits. Sheep, asses, and horses react to streptococcic infections. Spontaneous infections occur in these animals.

Little is known of the toxins produced by streptococci. In experimental work the secretion products of streptococci, as well as their protoplasmic toxins, are active only when used in large amounts (von Lingelsheim, Aronson). The formation of toxins is favored when suitable culture media are used (according to Marmorek, bouillon with the addition of leucin and glycocoll). [" G. F. Ruediger has shown that virulent streptococci produce a hæmolytic toxin, when grown in various heated serums, and has proved that this hæmolysin (streptocolysin) is a true toxin, possessing a haptophorous and toxophorous structure."—Ricketts' " Infection, Immunity, and Serum Therapy," p. 353.]

The blood serum of an animal immunized against streptococci (rabbit, mouse, ass, horse) protects other animals against infections which ordinarily prove fatal (Roger, Knorr, Marmorek, von Lingelsheim, and others). Streptococci from animals are not pathogenic (active) for man, and it is questionable whether immune sera from animals, even if the streptococci are taken from man, will be active. In the experiments of Koch and Petruschky prophylactic injections did not prevent the

development of erysipelas. The Tavel serum is taken from a horse which has been inoculated with forty-three strains of streptococci which were taken from man only and not passed through other animals. Favorable action is to be expected in general infections only when the serum is used in the beginning; in severe and old infections the endotoxin liberated by bacteriolysis causes a dangerous aggravation of the symptoms.

The blood serum of immunized animals possesses also agglutinating properties which are most active against those cocci with the cultures of which the animal has been immunized. Antilysins which neutralize the hæmolysins are likewise present in immune serum.

LITERATURE.—HAND AND TEXTBOOKS.—*Flügge.* Die Mikroorganismen (Frosch and Kruse). Leipzig, Vogel, 1896.—*C. Fränkel.* Bakterienkunde. Berlin, Hirschwald.—*Günther.* Bakteriologie. Leipzig, Thieme, 1902.—*Heim.* Bakteriologie. Stuttgart, Enke, 1898.—*Kolle* und *Wassermann.* Handb. d. pathogenen Mikroorganismen. Jena, 1903–4.—*Beitzke.* Ueber Agglutination der Staphylokokken durch menschliche Sera. Verhandl. d. pathol. Gesellsch., September, 1904. Zentralbl. f. allg. Pathol., Bd. 15, Ergänzungsheft, p. 154.—*Bruck, Michaelis* und *Schultze.* Beiträge zur Serodiagnostik der Staphylokokkenerkrankungen beim Menschen. Zeitschr. f. Hygiene u. Infektionskrankheiten, Bd. 50, 1905, p. 144.—*Fehleisen.* Zur Aetiologie der Eiterung. Arch. f. klin. Chir., Bd. 36, 1887, p. 966.—*E. Fränkel.* Ueber menschenpathogene Streptokokken. Münchner med. Woch., 1905, p. 1868.—*Fröhner* u. *Kärnbach.* Ein Beitrag zur primären infekt. Osteomyelitis des Pferdes. Monatsh. f. prakt. Tierheilkunde, Bd. 14, 1903, p. 433.—*Fromme.* Ueber prophyl. u. therap. Anwendung des Antistrept. Serums. Münch. med. Wochenschr., 1906, p. 20.—*Hentschel.* Pyämie und Sepsis. Festschr. f. Benno Schmitt. Leipzig, 1896.—*Jacobitz.* Ein Fall von Sepsis, hervorgerufen durch Staphylococcus citreus. Münchner med. Woch., 1905, p. 2020.—*Kerner.* Exp. Beitrag zur Hämolyse und zur Agglutination d. Streptokokken. Zentralbl. f. Bakteriol., Bd. 38, Orig., 1905, p. 223.—*Koch* und *Petruschky.* Beobachtungen über Erysipelimpfungen am Menschen. Zeitschr. f. Hygiene, Bd. 23, 1896, p. 477.—*Kolle* und *Otto.* Die Differenzierung der Staphylokokken mittelst Agglutination. Ibid., Bd. 41, 1902, p. 369.—*Lannelongue et Achard.* Étude exp. des Ostéomyélites à staph. et à strept. Annales de l'Inst. Pasteur, 1891, No. 4, p. 209.—*Lexer.* Experimente über Osteomyelitis. Arch. f. klin. Chir., Bd. 53, 1897, p. 266;—Die Schleimhaut des Rachens als Eingangspforte pyogener Infektionen. Ibid., Bd. 54, 1897, p. 736.—*v. Lingelsheim.* Streptokokken. In Kolle-Wassermanns Handb. d. pathog. Mikroorg., Bd. 3, 1903, p. 302;—Streptokokkenimmunität. Ibid., Bd. 4, 1904, p. 1186.—*Lubarsch.* Streptokokken als Krankheitserreger. Ergebn. d. allgem. Pathol. v. Lubarsch und Ostertag, January 3, 1896.—*Marmorck.* Die Arteinheit der für den Menschen pathogenen Streptokokken. Berl. klin. Wochenschr., 1902, p. 299;—Das Streptokokkengift. Ibid., p. 253.—*Fritz Meyer.* Die klin. Anwendung des Strept.-Serums. Zentralbl. f. Bakt., Bd. 36, Refer., 1905, p. 309.—*Miller.* Die Mikroorganismen der Mundhöhle. Leipzig, 1892.—*v. Mikulicz.* Die neuesten Bestrebungen der aseptischen Wundbehandl. Chir.-Kongr. Verhandl., 1898, II, p. 1.—*Natvig.* Bakt. Verhältnisse in weibl. Genitalsekreten. Arch. f. Gynäkol., Bd. 76, 1905, p. 701.—*Neisser* und *Lipstein.* Die Staphylokokken. In Kolle-Wassermanns Handb. d. pathog. Mikroorg., Bd. 3, 1903, p. 105.—*Neisser.* Staphylokokkenimmunität. Ibid., Bd. 4, 1904, p. 1150.—*Neisser* and *Wechsberg.* Ueber das Staphylotoxin. Zeitschr. f. Hygiene, Bd. 36, 1901, p. 299.—*Passet.* Untersuchungen über die Aetiologie der eitrigen Phlegmone des Menschen. Berlin, 1885.—*Petruschky.* Untersuchungen

über Infektion mit pyogenen Kokken. Zeitschr. f. Hygiene, Bd. 17, 1894, p. 59;—Entscheidungsversuche zur Frage der Spezifität der Erysipelstreptokokken. Ebenda, Bd. 23, 1896, p. 142;—Ueber die Konservierung virulenter Streptokokkenkulturen. Zentralbl. f. Bakteriolog., Bd. 17, 1895, p. 560.—*Pröscher.* Die Gewinnung von Antistaphylokokkenserum. Ibid., Bd. 37, Orig. 1904, p. 295.—*Rodet.* De la nature d'ostéomyélite infectieuse. Comptes rendus de l'académie des sciences, 1884.—*Rosenbach.* Mikroorganismen bei den Wundinfektionskrankheiten des Menschen. Wiesbaden, 1884.—*Schottmüller.* Die Artunterscheidung der für den Menschen pathogenen Streptokokken durch Blutagar. Münchn. med. Woch., 1903, p. 849.—*Tavel.* Experim. u. Klin. über das polyvalente Antistreptokokkenserum. Deutsche med. Wochenschr., 1903, p. 950.

(c) DIPLOCOCCUS PNEUMONIÆ

The diplococcus pneumoniæ, diplococcus or streptococcus lanceolatus, pneumococcus was demonstrated by A. Fraenkel in 1886 to be the cause of croupous pneumonia in man, and his findings were later confirmed by Weichselbaum. The same micro-organism had been found in rabbits dying of a general fatal infection (so-called sputum-septicæmia) following the injection of human saliva by Pasteur in 1881, and had been found by Rosenbach in 1884 and described as the micrococcus pyogenes tenuis. Gradually it has been recognized that the pneumococcus may be the cause of different inflammations following or occurring independent of pneumonia.

The separate halves of the diplococcus are shaped like a lancet or candle flame. The pneumococcus is not motile. It possesses a capsule which is constantly present when the bacteria are found in the tissues, in the blood of man and animals, and which may be present when the pneumococcus is grown on certain culture media (milk and serum). The capsule appears pale when the ordinary stains are employed (aniline stains and Gram's method) (Fig. 91).

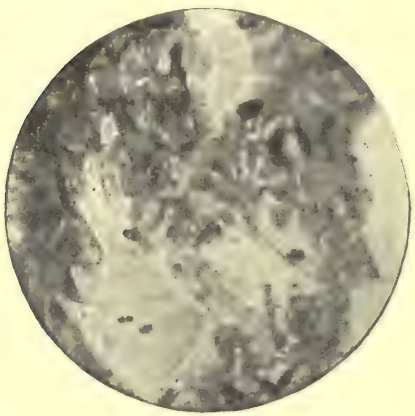


Fig. 91.

It may be cultivated most easily upon slightly alkaline culture media at high temperatures. Upon agar and blood serum the cultures appear as small transparent drops resembling closely streptococcic colonies. Gelatin which is not liquefied is less suited for a culture medium (at 22° to 24° C.). Bouillon is somewhat clouded in the first few days.

Pneumococci die rapidly upon all culture media, and must therefore be transplanted daily. When transplanted they may undergo a number of changes; sometimes the individual pneumococci become more round, sometimes more oval, the capsule may be wanting, and frequently they form long chains. All transitions to the form of the streptococcus pyogenes may be obtained by growth upon culture media (Kruse and Pansini), yet the original type may be obtained if they are passed through animals a number of times. [" Recently the danger of confusing the pneumococcus with the streptococcus has received renewed attention, and newer methods of differentiation render it extremely probable that such confusion has occurred in the past. An important differential method is that of cultivation on agar plates which contain blood (Schottmueller); the streptococcus produces a clear zone of hæmolyzed corpuscles about its colonies, whereas the colonies of pneumococcus present a greenish color and produce no hæmolysis."—Ricketts' " Infection, Immunity, and Serum Therapy," p. 338.]

Pneumococci remain viable and virulent for a long time in the dried expectoration of pneumonia patients.

Pneumococci occur most frequently in fibrinous pneumonia, of which, according to Weichselbaum, they are the cause in seventy-one per cent of the cases, and in catarrhal broncho-pneumonia. Pleurisy, peritonitis, endopericarditis, arthritis, osteomyelitis, suppuration in soft tissues, and viscera, and the puerperal uterus may occur secondary to pneumococcic infection of the lungs. Infection in these cases occurs through the blood, except where pleurisy or peritonitis develops as the result of direct extension. Infections may occur independently of any inflammation of the lung, as pneumococci occasionally gain access to wounds and cause severe inflammations (e. g., mastitis, Green) or phlegmons which may end in general infection, with or without metastatic suppuration. Frequently they extend from the buccal and nasal cavities, in which they are found in an attenuated condition in healthy individuals (Sanarelli, Weichselbaum), and produce suppuration of the accessory sinuses (frontal and maxillary sinuses), otitis media, and parotitis. Pneumococci, carried by the saliva or food into the stomach, may cause peritonitis if the mucous membrane is ulcerated or inflamed (Weichselbaum, de Quervain). Hæmatogenous infection of the peritoneum after pneumonia is rare (Jensen).

That the virulence of pneumococci changes easily is indicated by the different forms of inflammation (serous, fibrinous, and suppurative) which it causes. In animal experiments it is not difficult to demonstrate that pneumococci from different sources differ in virulence, and the important fact that in the course of pneumonia the cocci become attenuated as the crisis approaches.

Rabbits and mice, the most susceptible animals, die after subcutaneous injections of small amounts of pneumococcic sputum or cultures in from one to two days of a general infection without local or metastatic suppuration. Pneumococci are then found in large numbers in the blood, serous cavities, viscera, spleen and bone marrow. The more attenuated the pneumococci injected, the more frequently local inflammation, suppuration, even erysipelas (Neufeld), and metastatic foci (pneumonic infiltration, suppuration in joints) develop and the longer the animals remain alive. Nothing definite is known concerning the formation of toxins; apparently an endotoxin is produced.

The blood serum of patients convalescing from pneumonia contains, according to F. and G. Klemperer, Huber, and Blumenthal, specific bactericidal bodies which are formed, according to A. Wassermann, mostly in bone marrow. Blood serum from convalescents also agglutinates pneumococci. The blood serum of horses, immunized against pneumococci, prevents and cures infections in other animals (Mennes and others). The antipneumococcic serum is of no practical importance as yet.

LITERATURE.—*A. Fränkel.* Ueber Pneumokokkenbefunde im Blute bei der menschl. Lungenentzündung. Internat. Beitr. z. inn. Med., II, 1902.—*Green.* The bacteriology of mastoiditis. Zentralbl. f. Bakteriol., Bd. 30, 1901, p. 468.—*Ilentschel.* Beitrag zur Lehre von der Pyämie u. Sepsis. Festschr. f. Benno Schmitt, Leipzig, 1896.—*Huber* und *Blumenthal.* Ueber die antitoxische u. therap. Wirkung des menschl. Blutes nach überstandenen Infektionskrankheiten. Berlin. klin. Wochenschr., 1897, p. 671.—*Jensen.* Ueber Pneumokokkenperitonitis. Arch. f. klin. Chir., Bd. 70, 1903, p. 91.—*G.* und *F. Klemperer.* Versuche über Immunisierung und Heilung bei der Pneumokokkeninfektion. Berl. klin. Wochenschr., 1891, p. 833.—*v. Leyden.* Pneumonie. Die Deutsche Klinik, Bd. 2, 1903. Serumtherapie, p. 298.—*Menetrier* et *Aubertin.* Péritonite à pneumocoques. Soc. méd. des hôpitaux, Paris, 1901.—*Mennes.* Das Antipneumokokkenserum u. s. w. Zeitschr. f. Hygiene, Bd. 25, 1898, p. 413.—*de Quervain.* Zur Aetiologie der Pneumokokkenperitonitis. Korrespondenzbl. f. Schweizer Aerzte, 1902.—*Sanarelli.* Der menschl. Speichel und die Mikroorganismen der Mundhöhle. Zentralbl. f. Bakteriol., Bd. 10, 1891, p. 817.—*Schabad.* Ein Fall von allgemeiner Pneumokokkeninfektion. Zentralbl. f. Bakteriol., Bd. 19, 1896, p. 991.—*M. Wassermann.* Pneumokokkenschutzstoffe. Deutsche med. Wochenschr., 1899, p. 141.—*Weichselbaum.* Diplococcus pneumoniae. In Kolle-Wassermanns Handb. der pathog. Microorg., Bd. 3, p. 189, 1903 mit Lit.—*Ders.* Pneumokokkenimmunität. Ibid., Bd. 4, 1904, p. 1164.

(d) MICROCOCCUS TETRAGENUS

The micrococcus tetragenus was first described by Koch (1884) and Gaffky. It may be easily recognized morphologically, as four cocci are grouped within a capsule. This micrococcus is found in the pus of tuberculous lung cavities, and occurs quite frequently in human sputum (Biondi, Sanarelli). It is found more rarely in abscesses and phlegmons of the neck.

It grows upon gelatin (without liquefaction) and upon agar (forming white or grayish yellow colonies), and stains with aniline dyes and by Gram's method. White mice and guinea pigs die after subcutaneous injections of a general bacterial infection.

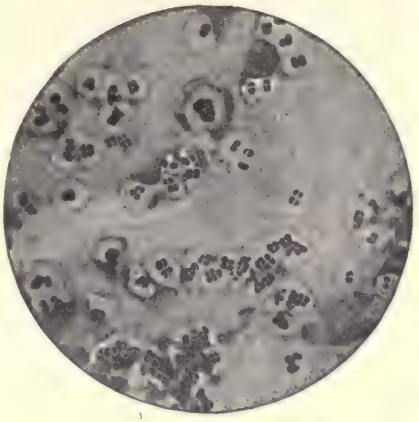


FIG. 92.

LITERATURE.—*Sanarelli.* Der menschliche Speichel und die Mikroorganismen der Mundhöhle. Zentralbl. für Bakteriol., Bd. 10, 1891, p. 817.

(e) MICROCOCCUS GONORRHŒÆ, GONOCOCCUS

The gonococcus was first found by Neisser (1879) in gonorrheal pus, and cultivated upon human blood serum by Bumm (1885). Its specific relation to gonorrhea was demonstrated by the successful inoculation of pure cultures into the urethra of man.

It is a diplococcus. The broad and hilus-like side of the individual cocci, which are of a hemispherical, coffee-bean, or reniform shape, face each other. In pus they occur in groups, and are either free or inclosed within the cytoplasm of the cells lying close to the nucleus (Fig. 93).

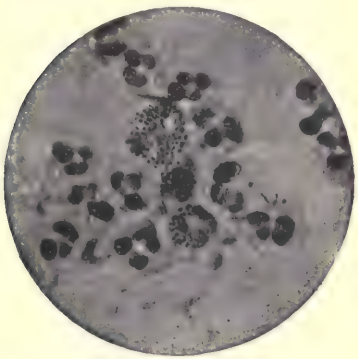


FIG. 93.

They stain with aniline dyes (best with carbolfuchsin and methylene blue) and are destained by Gram's method. They may be cultivated most easily upon agar, the surface of which has been thickly covered with human blood (Abel), or upon the culture media (peptonagar with nutrose and glycerin agar) recommended by A. Wassermann. They do not grow upon the ordinary media. The temperature most favorable for growth is about 36° C.; growth ceases at temperature over 38° C. The whitish gray colonies must be examined carefully microscopically, for there is nothing characteristic about their macroscopic appearance. In making a diagnosis, not only the form of the cocci, but their growth upon special and ordinary culture media, and the way in which they react the Gram's stain, must be considered.

Gonococci are so sensitive to drying that they are killed after twenty-four hours; on the other hand, they resist the ordinary agents used in the treatment of gonorrhea for from five to ten minutes (Bumm).

Animals show but little susceptibility, their urethra and conjunctiva remaining healthy after being inoculated with gonococci. White mice and guinea pigs either develop a circumscribed suppurative peritonitis after artificial infection of their peritoneum, or die after twenty-four hours without any special local inflammation. The cocci die rapidly in the animal's body, and so do not produce general infections. They act through their toxins, and usually produce only a local reaction. The joint infections produced by living or dead cultures are benign, and the suppuration ceases spontaneously (Nicolaysen).

According to A. Wassermann and Nicolaysen, the active toxins are united with the protoplasm, and are only set free when the cocci die. De Christmas is the only one who has found a soluble toxin which is developed in the culture media. Experimentally, immunization against the toxin is possible.

The field of action of the gonococcus in man is extensive. Periurethral abscesses, epididymitis, prostatitis, cystitis, ureteritis, pyelonephritis may develop secondarily to a gonorrhea in man. The gonococcus plays even a greater rôle in the pathology of the female genital organs. Secondary to a gonorrhea of the urethra or cervix, endoparametritis, salpingitis, oöphoritis, peritonitis, cystitis, pyelitis, and proctitis may develop. In the newborn the gonococcus may produce the dangerous ophthalmia neonatorum and relatively frequently the stomatitis gonorrhoica.

All these inflammations are for the greater part suppurative in character. If the tendency to spread, as seen in staphylo- and streptococcic suppuration, is absent and therefore the course of the infection usually benign, often enough they cause severe and even fatal infections. The gonococci extend along mucous membranes and through lymphatic channels, and not infrequently gain access to blood vessels. They do not always produce a general infection, however, as they remain but a short time in the blood, being deposited, as has been demonstrated many times (Proschaska), in different parts of the body, even upon the valves of the heart. They have been found in fatal endocarditis and in all possible forms of metastatic serous, serofibrinous, and suppurative inflammation, such as abscesses of soft tissues, tendovaginitis, bursitis, pleuritis, arthritis, myocarditis, once even in perichondritis (Finger), in osteomyelitis (Ullmann) and parotitis (Colombini). The pure gonococcic metastatic inflammation is characterized by its benignity, notwithstanding its acute febrile onset. The gonococci soon die, but the serous exudation into the surrounding tissues, the pain, and the contractures remain for a

long time. The suppurative peritonitis with acute severe onset tends to become encapsulated and may heal spontaneously.

The severe progressive inflammations and metastatic processes are, as a rule, caused by the staphylococci and streptococci, as frequently the gonorrheal inflammation of the mucous membranes provides an infection atrium for these bacteria.

A previous gonorrheal infection does not protect against a later infection.

LITERATURE.—*Abel.* Zur Gonokokkenkultur. Deutsche med. Wochenschr., 1893, p. 265.—*E. Bumm.* Die gonorrh. Erkrankungen der weibl. Harn- und Geschlechtsorgane Handb. d. Gynäk. von Veit, Bd. 1.—*de Christmas.* Contrib. à l'étude du Gonocoque et de sa toxine. Annales de l'Inst. Pasteur, 1900, No. 5, p. 331.—*Colombini.* Untersuchungen über einen Fall von allgem. gonorrh. Infektion. Zentralbl. f. Bakteriol., Bd. 24, 1898, p. 955.—*Councilman.* Gonorrhœal myocarditis. The American Journal of the Med. Sciences, Bd. 106, 1893.—*Finger.* Die Gonokokkenpyämie. Wiener klin. Wochenschr., 1896.—*Finger, Ghon* und *Schlagenhaufer.* Beitr. zur Biologie des Gonokokkus. Arch. f. Dermat. u. Syphilis, Bd. 28, 1894, p. 277;—Endocarditis, Arthritis, Prostatitis. Ibid., Bd. 33, 1895, p. 141.—*v. Hofmann.* Gonorrhoische Allgemeininfektion und Metastasen. Zentralbl. f. Grenzgeb., Bd. 6, 1903, p. 242.—*Jacobi* und *Goldmann.* Tendovaginitis suppurativa gonorrhoica. Beitr. z. klin. Chir., Bd. 12, 1894, p. 827.—*Michaelis.* Ueber Endocarditis gonorrh. und andere gon. Metastasen. Internat. Beitr. zur inner. Med., Bd. 2, 1902.—*Neusser* und *Scholtz.* Gonorrhoe. In Kolle-Wassermanns Handb. der path. Mikroorganismen, Bd. 3, 1903, p. 148.—*Nicolaysen.* Zur Pathogenität und Giftigkeit des Gonokokkus. Zentralbl. f. Bakteriol., Bd. 22, 1897, p. 305.—*Proschaska.* Ueber die gonorrh. Allgemein infektionen. Virch. Arch., Bd. 164, 1901, p. 494.—*Scholtz.* Immunität bei Gonorrhoe. In Kolle-Wassermanns Handb. d. pathog. Mikroorg., Bd. 4, 1904, p. 110.—*Ullmann.* Osteomyelitis gonorrh. Wien. med. Presse, 1900, No. 49;—Ueber Allgemeininfektion nach Gonorrhoe. Deutsch. Arch. f. klin. Med., Bd. 69, 1901, p. 309.—*Unger.* Gonokokken im Blute bei gon. Polyarthritis. Deutsche med. Wochenschr., 1901, p. 894.—*A. Wassermann.* Ueber Gonokokkenkultur und Gonokokkengift. Berl. klin. Wochenschr., 1897, p. 685;—Ein durch Gelingen der Reinkultur bewiesener Fall von Endocarditis gonorrh. Münch. med. Wochenschr., 1901, p. 298

(f) BACILLUS PYOCYANEUS

Not infrequently dressings saturated with wound secretion have a greenish or bluish color and a characteristic sweetish odor. The bacteria causing this color and odor were discovered by Luecke in 1862; pure cultures were first obtained by Gessard in 1882. They have been called the bacilli of green or blue pus; the pus itself, however, is not colored, but the dressings, the clothes, and the skin surrounding the wound assume the greenish or bluish color when moistened with secretion. The deeper layers of large dressings have a yellowish or brown color, as insufficient amounts of air reach the secretion to give rise to the blue color.

The bacillus is a small, actively motile rod which does not form spores. It stains with aniline dyes, and is destained by Gram's method.

They form whitish colonies and deposits upon solid culture media, which soon assume a deep bluish green color and develop a peculiar odor. Bouillon cultures also assume this color and odor. Gelatin is liquefied.

The color of the cultures is produced by two coloring matters which develop throughout the culture when the bacilli are not altered by growth upon poor culture media, and when the culture medium is suitable and there is free access of air. Old cultures upon poor media lose their ability to produce these coloring matters. Vigorous bacilli cannot form these even upon good culture media, when there is not sufficient oxygen and the media do not contain glucose. When symbiotic with other bacteria, such as the staphylococci and streptococci, no coloring matter is produced (Muehsam and Schimmelbusch, Paul Krause).

One of the coloring matters (pyocyanin) may be extracted from cultures or dressings with chloroform, and when the chloroform evaporates it crystallizes out in the form of long blue needles. The other coloring matter (pyofluorescin) is not soluble in chloroform, is green, and fluorescent. It depends upon the character of culture medium whether one or the other develops alone or in excess. According to Gessard pyocyanin alone is formed in pure peptone solutions; pyofluorescin alone in uncooked egg albumen.

The bacillus pyocyaneus is frequently found in the human skin, and may be easily demonstrated in areas (inguinal fold, axillary fossa, and crena ani) which are well supplied with sweat glands (Muehsam). It may be made to grow actively, as indicated by the development of the green color, by the use of moist warm compresses. The so-called green sweat, which stains the clothing of many individuals, is produced by the growth of the bacillus pyocyaneus (Eberth). If the bacilli once settle upon a part of the body, or their development is favored by the use of warm compresses for a week, it is impossible to remove all of the bacteria before an operation, even by the most careful sterilization, and in a few days the dressings will be stained green. The presence of the bacilli in the skin, and their resistance to antiseptic solution explain satisfactorily their frequent occurrence in wound infections. There is not much danger of transferring these bacilli by the hands during operations or change of dressings.

The bacillus pyocyaneus is almost never absent in chronic suppuration about a fistula. It occasionally occurs as a saprophyte in the intestinal contents, in the bladder, and urethra.

Its presence in the skin about sutured wounds, or even in the wound itself, does not disturb to any extent wound repair, as it usually causes merely mild suppuration of stitch holes with some elevation of temperature; rarely necrosis of connective tissue and muscles. Open, granulating

wounds suffer much more from infection with the bacillus pyocyaneus. Even in these cases the bacilli do not pass into the deeper tissues, causing suppurative inflammation accompanied by fever, but excite a profuse wound secretion which interferes with wound repair and forms a fibrinous coating which covers the granulating surface and retards the growth of epithelium over it. Skin grafting of such wounds is never successful.

Occasionally in man the bacillus pyocyaneus becomes both pathogenic and pyogenic. Clinical observations of cases in which the bacillus is pathogenic are reported frequently, but only a few of these can be accepted without reserve. Green found it alone in eight cases of mastitis. Dangerous, even fatal, pyocyaneus infections may develop in nursing children (Kossel, M. Wassermann). It has been found in otitis media, meningitis, enteritis, and suppurative thrombosis of the umbilical arteries with metastatic foci in the lungs. In many cases of the latter which were apparently epidemic and presented the clinical picture of a general infection, M. Wassermann cultivated a virulent pyocyaneus bacillus from the different foci. The bacillus pyocyaneus has been frequently found in the blood of small children dying of an enteritis accompanied by fever and hæmorrhages.

In animals (guinea pigs and rabbits) the bacillus produces a powerful toxin, although it does not multiply rapidly. It produces no disturbance in wounds, but after subcutaneous or intravenous injection of small amounts of a virulent culture a severe infection (with nephritis and hæmorrhages into gastrointestinal mucous membrane) develops which proves fatal in twenty-four hours or after many weeks. Death occurs rapidly after the injection of larger doses, and bacilli can be demonstrated in the blood. Bacilli are not found in the blood, however, when smaller doses are injected, and the animal lives for some time, as the bacilli are excreted by the liver and kidneys and can be demonstrated in the bile and urine. They appear in the latter in a quarter of an hour after injection (von Klecki). Paralysis and degeneration of the viscera develop in chronic cases.

Large enough doses of sterile cultures have a similar action to that described above, as they contain toxins secreted by the bacilli. The toxins must not be considered as identical with the coloring matters. In man the injection of sterile cultures produces a general (mild fever) and local (erysipelatous) reaction (Schimmelbusch). The poisons are partly toxins which are held in solution in the culture fluids, and partly endotoxins which are bound to the bacterial protoplasm. The endotoxin dissolves red blood corpuscles, and therefore contains a hæmolytic poison or pyocyanolysin (Bullock and Hunter). It is questionable, however, whether this hæmolytic action is not due to the alkali content of the culture medium (Jordan).

Animals may be artificially immunized. A. Wassermann has shown that the blood serum of animals immunized with the soluble toxin is active against the bacilli and the toxin. The serum is therefore both bactericidal and antitoxic. The serum of animals immunized with living cultures has only a bactericidal action.

(g) BACTERIUM COLI COMMUNE

This bacillus was found by Emmerich (1884) in patients dying of cholera, and was regarded by him as the cause of the disease. Later (1885) it was demonstrated by von Escherich to be one of the ordinary intestinal bacteria of nursing children, and was apparently cultivated simultaneously by Passet from the pus of a periprocteal abscess (bacillus pyogenes fœtidus).

They occur in the form of short rods, singly or in pairs, among which coccoid forms may be found; are provided with flagella, and are therefore capable of motion. The bacilli do not form spores; are fairly resistant to high and low temperatures, but not to drying (Walliczek); stain with the aniline dyes, but are destained by Gram's method.

They form upon gelatin small, round, yellowish white colonies. Gelatin is not liquefied. Stab cultures resemble a nail with a flat head as the bacilli develop rapidly upon the surface of the gelatin. The bacilli form transparent, grayish white deposits upon agar, and render bouillon very cloudy. In glycerin and media containing grape or milk sugar they produce acid fermentation and gas. The formation of gases (H and CO_2) may be most easily demonstrated in stab cultures in glucose agar, in which numerous gas bubbles develop in twenty-four hours.

The formation of acids (lactic, acetic, and formic) may be easily demonstrated by growth in sterile milk, which becomes curdled after a few days. They produce in sugar free but peptone-containing media, to which has been added potassium nitrite and sulphuric acid, a red coloring substance (nitroso-indol). (This test may be used to differentiate the colon from other closely related bacteria.)

The bacillus coli communis is a very widely distributed saprophyte. It plays an important rôle in pathology, both as a pyogenic and putrefactive bacterium. It inhabits with closely related species (the groups of colon bacilli) the intestine, especially the large intestine of man and many animals, but also occurs outside of the body (e. g., in water, air, in the dust of schoolrooms, upon the skin, in the clothing, etc.). Its presence in the intestinal contents becomes of significance only in pathological conditions. In cholera, dysentery, typhoid fever, cholera nostrans, and the common forms of enteritis, an infection atrium is provided for the colon bacilli which frequently increase rapidly in numbers and

virulence. As a result of this increase in virulence following lesions in the intestines, they may produce peritonitis after passing through the intestinal wall or after absorption, metastatic suppuration, or general infection. Infection atria are provided for colon bacilli by wounds of the intestinal mucous membranes, by intestinal perforation following ulcer or injury, by circulatory disturbances and invaginations which render the intestinal wall more permeable for bacteria. Colon bacilli may cause circumscribed, progressive, or general suppurative peritonitis. They are frequently found in appendiceal and periprocteal abscesses, in suppurative cholecystitis and cholangitis, and in abscesses of the liver following these lesions, and occur also in general bacterial infections and metastatic foci (endocarditis, pleuritis, meningitis, arthritis, periostitis, etc.).

It is conceivable that in severe intestinal catarrh the bacilli may be absorbed and pass into the lymphatic and blood vessels (Seitz). It is doubtful, however, whether in all cases of meningitis, pleuritis, and strumitis in which the colon bacillus is found the infection occurred in this way, as the bacilli are widely distributed in the external world and the infection may have occurred from without. The colon bacillus has been found in felons, in lymphangitis ending in suppuration, often with the formation of gas (gas phlegmon), in otitis media, and in a few cases of osteomyelitis in association with other bacteria. Recent operation-wounds infected with the colon bacillus are foul smelling and dry and are accompanied by an elevation of temperature. The wound surfaces become necrotic and gangrenous, and granulation tissue which secretes profusely is formed, but slowly (cf. Putrefactive Infections).

If the bacilli gain access to the bladder, this occurring most frequently in man during catheterization, severe infections (cystitis, pyelitis, pyelonephritis) which may prove fatal if they become general may develop. Sittmann and Barlow in such a case cultivated the bacillus coli communis from the blood eleven hours before death. If the bacillus gains access to the vagina, puerperal infections may develop (Eisenhart).

The demonstration of colon bacilli in cadavers is no proof that they had any causal connection with death, for the bacilli pass through the intestinal wall shortly after death and become widely distributed.

The mouse, guinea pig, rabbit, dog, and cat are best suited for experimental purposes. Abscesses follow cutaneous and subcutaneous injections. After intraperitoneal and intravenous injections of virulent cultures, there develops besides the suppurative peritonitis following intraperitoneal injection a fatal general infection, associated almost always with a severe enteritis, bacteria occurring in the blood and viscera. Ackermann produced osteomyelitis in young animals by intravenous injections. Guyon produced a cystitis by intravenous injections, having

caused previously a urinary retention by closing the urethra. According to Buchner the pyogenic action depends upon chemotactic substances in the protoplasm, but the culture also contains soluble toxins.

A susceptible animal may be immunized by injecting increasing doses of living cultures, and the blood serum obtained from immunized animals will agglutinate the bacilli (*vide* Typhoid Bacilli; cf. also Putrid Infections).

LITERATURE.—*Ackermann.* Les ostéomyélites exp. prov. par bact. coli comm. Arch. de méd. exp. T. 7, 1895, p. 330.—*Brunner.* Eine Beobachtung v. Wundinfektion durch d. Bact. coli comm. Zentralbl. f. Bakteriol., Bd. 16, 1894, p. 993.—*Cacace.* Die Bakterien d. Schule. Zentralbl. f. Bakteriol., Bd. 30, 1901, p. 653.—*Dmochowski* und *Janowski.* Zwei Fälle von eiteriger Entzünd. der Gallengänge durch Bact. coli comm. Zentralbl. f. allgem. Pathol., Bd. 5, 1894, p. 277.—*Eisenhart.* Puerperale Infektion m. tödl. Ausgange durch Bact. coli comm. Arch. f. Gyn., Bd. 47, 1894, p. 189.—*Escherich* u. *Pfaundler.* Bact. coli comm. In Kolle-Wassermanns Handb. der pathog. Mikroorganismen, Bd. 2, 1903, p. 334.—*Gibbert.* De la colibacillose. Semaine méd., 1895.—*Henke.* Beitrag zur Verbreitung des Bact. coli comm. in der Aussenwelt. Zentralbl. f. Bakteriol., Bd. 16, p. 481, 1894.—*Hitschmann* u. *Michel.* Eine vom Bact. coli comm. hervorgerufene Endokarditis u. Pyämie. Wien. klin. Wochenschr., 1896.—*Jatta.* Experim. Untersuchungen über die Agglutination d. Typhusbaz. u. d. Mikroorg. aus d. Koligruppe. Zeitschr. f. Hygiene, Bd. 33, 1900, p. 185.—*Kiessling.* Das Bact. coli comm. Hygien. Rundschau, Bd. 3, 1893, p. 724.—*Krogius.* Ueber den gewöhnl. bei der Harninfektion wirksamen path. Baz. (Bact. coli comm.). Zentralbl. f. Bakteriol., Bd. 16, 1894, p. 1006.—*Oker-Blom.* Beitrag zur Kenntnis des Eindringens des Bact. coli comm. in die Darmwand in path. Zuständen. Zentralbl. f. Bakt., Bd. 15, 1894, p. 588.—*Pfaundler.* Spez. Immunitätslehre betr. Bact. coli. In Kolle-Wassermanns Handb. d. pathog. Mikroorg., Bd. 4, 1904, p. 905.—*Seitz.* Darmbakterien und Darmbakteriengifte im Gehirn. Korresp.-Bl. f. Schweiz. Aerzte, 1900.—*Sittmann* und *Barlow.* Ueber einen Befund von Bact. coli comm. im lebenden Blut. Deutsch. Arch. f. klin. Med., Bd. 52, 1894, p. 250.—*Walliczek.* Resistenz des Bact. coli comm. gegen Eintrocknen. Zentralbl. f. Bakteriol., Bd. 15, 1894, p. 949.

(h) BACILLUS TYPHOSUS

The typhoid bacillus was first seen and described by Eberth (1880) in the viscera of patients dying of typhoid fever; later by Koch. Pure cultures of the bacillus were first obtained by Gaffky (1884).

The bacilli are short and thick rods with rounded ends which do not form spores. They are provided with long, actively motile, wavy flagella, which are attached all along the sides and at ends of the bacilli.

The typhoid bacilli grow upon the ordinary media, best when there is free access of air. In gelatin, which is not liquefied, the superficial colonies are flat, with wavy margins, while the deep colonies are about the size of the head of a pin, round and grayish white in color. In stab cultures white threads are formed. Upon agar they form a transparent, closely attached film. Bouillon is clouded rapidly.

The typhoid bacilli have a number of characteristics which enable one to differentiate them from the colon bacillus and closely related forms. They do not form gas in culture media containing glucose, do not coagulate milk, and produce no color reaction (Indol) (*vide* Bacterium Coli Commune). No single test is sufficient to distinguish the typhoid from the colon bacillus; a number must be tried (see below, Pfeiffer, Widal).

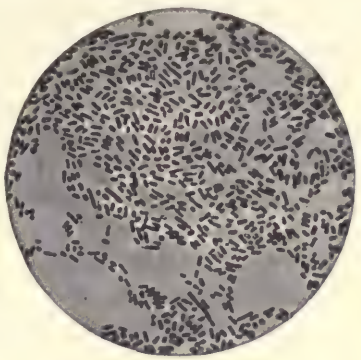


Fig. 94.

In moist conditions the bacillus is viable for a long time, in water some weeks, in fecal masses many months (Uffelmann, Karlinski); on the other hand, it is rapidly destroyed by drying (Kruse).

Typhoid bacilli are excreted in the feces and urine; in the latter even during convalescence (Petruschky). They remain viable for a long time outside the body in damp places (e. g., the floor), so that the danger of infection is great. The bacilli may be easily transferred to the mouth by the infected fingers or in drinking water, during an epidemic, which comes from wells adjacent to privies or outhouses.

The intestinal canal affords the infection atrium for the typhoid bacilli. They settle in the lymph follicles and cause inflammation, necrosis, and ulceration. Nothing is known of the symptoms of wound infections with typhoid bacilli.

The bacilli do not remain confined to the lymph follicles in the intestinal wall and mesenteric lymph nodes, which are soon involved. They early pass into the blood stream and become distributed in small foci in all the viscera and tissues, and during pregnancy may even be deposited in the fœtus. They remain only temporarily in the blood, occurring in the greatest numbers during the eruptive stage, and therefore there may be some difficulty in demonstrating them in the blood, although it has frequently been done (Castellani and Schottmueller, Burdach, and others). The bacilli are found most abundantly in the spleen, then in the bone marrow and periosteum; they have also been demonstrated in the liver and gall bladder,[1] in the kidney, the rose spots, and in the heart valves in endocarditis. Groups of bacilli may remain in the body for a long time without giving rise to symptoms; for example, Buschke has

[1] Infection of the mucous membrane of the gall bladder with typhoid bacilli is frequently followed by a chronic catarrhal inflammation, which is an etiological factor in gall-stone formation.

found them in an osteal focus seven years after typhoid fever, Sultan six years after.

Occasionally, but relatively infrequently, they cause inflammation and suppuration, which occurs most frequently during convalescence (post-typhoid inflammation). If the typhoid bacilli act alone the inflammation usually pursues a mild and chronic course. The pus which is formed is thin, reddish yellow in color, and contains but few cells.

The pyogenic action of typhoid bacilli has been proven by animal experimentation, and by finding them unassociated with other bacteria in different forms of post-typhoid suppurative inflammation, such as suppuration of marrow of bone and periosteal foci with abscesses of soft tissues, in suppuration of the subcutaneous tissues, and of muscle, in arthritis and in suppurative foci in the viscera, parotid gland, goiter, eye, testicle, epididymis, ovary, spleen, liver, in pleurisy, meningitis, and peritonitis.

Quite frequently post-typhoid inflammations pursue a severe clinical course. In these cases pyogenic cocci and other micro-organisms (pneumococci, colon bacilli) are often the cause of the inflammation, the typhoid infection in the intestine providing the infection atrium or conditions favorable for the development of these bacteria. In these severe cases the typhoid bacilli have been found with other bacteria or the latter alone. It is possible, however, that in these cases the typhoid bacilli may have died in the abscesses or that the infection with pyogenic cocci was secondary.

The entire course of the disease may be influenced by a mixed infection, and that a general bacterial infection with other bacteria may develop has been positively demonstrated by finding these bacteria in the blood.

Guinea pigs and mice are the most susceptible of all the animals used for experimental purposes. They die in from one to two days after the injections of small amounts of typhoid cultures into the peritoneal cavity. Death is due to the action of the toxins, as the bacilli are found in the blood only after the use of much larger amounts, and are then deposited in the viscera. After the injection of slightly virulent cultures the animal lives for some days, and only a few bacteria can then be found. The experimental production of the disease, as it occurs in man, by the feeding of cultures, has been successful to only a limited degree.

The pyogenic action of the bacillus after injection into different tissues, joints, and body cavities of rabbits and dogs has been positively demonstrated (Orloff, Dmochowski and Janowski, Kruse).

The virulence of typhoid bacilli decreases upon culture media, but may be raised by passing them through animals.

The toxins are partly present in the culture media and partly freed by the death of the bacteria, both the culture filtrate freed of bacteria and the bacilli killed with chloroform vapor being active. The toxins

have a special action upon the intestinal mucous membrane (Sanarelli). In man sterile cultures injected in small amounts produce a rapid but transitory reaction (R. Pfeiffer and Kolle). According to E. and P. Levy the culture filtrate contains a hæmolytic substance, as it dissolves red blood corpuscles.

A susceptible animal may be immunized by one injection of a culture in which the bacilli have been killed (Brieger, Kitasato, and A. Wassermann).

According to R. Pfeiffer, the blood serum of an immunized animal is not antitoxic, but bactericidal. It kills and dissolves typhoid bacilli in a short time, but does not neutralize the toxins; the serum should therefore be used only as a prophylactic measure. The blood serum of convalescent patients and individuals who have been inoculated with small amounts of a sterile culture have this bactericidal property (Pfeiffer and Kolle). The lymph nodes, spleen, bone marrow, and thymus gland must be regarded as the principal sources of typhoid antibodies (Wassermann). Protective inoculation of soldiers with dead cultures has been performed with favorable results (Gaffky and Kolle).

In order to lessen the unpleasant symptoms following inoculation, which are apparently due to the decomposition products of the bacteria and the culture media, Bassenge and Mayer prepared a filtrate from typhoid cultures which was freed of the decomposition products by shaking in sterile water.

According to Pfeiffer, the specific bactericidal properties of the serum of artificially immunized animals may be used to establish the identity of questionable typhoid bacilli. In this test the bacilli in question should be injected into the peritoneal cavity of an immunized guinea pig. If the animal remains alive the bacilli injected are typhoid, as the bacilli are killed by the serum. The solution of the bacteria may be followed under the microscope if some of the peritoneal fluid is withdrawn with a capillary tube, and a hanging drop prepared. This reaction is specific if the animal is immunized against typhoid, and therefore can be used to establish the identity of the typhoid bacillus.

In vitro the typhoid immune serum, which within the body kills and dissolves typhoid bacilli, has another action. When a suspension of the bacilli are mixed with the serum upon a slide or in a small tube, they rapidly lose their motility and become agglutinated. This reaction depends upon substances which, because of their action, have been called specific agglutinins. The blood serum of many typhoid patients early shows this specific agglutinating property (Widal and Gruber), and this has been made use of in making a diagnosis of typhoid fever in the Widal or sero-diagnostic test. A negative result does not, however, exclude typhoid, for this agglutinating property of serum develops late in many cases.

LITERATURE.—*Bassenge* und *Mayer*. Zur Schutzimpfung gegen Typhus. Deutsche med. Wochenschr., 1905, p. 697.—*Brieger, Kitasato* und *Wassermann*. Ueber Immunität und Giftfestigung. Zeitschr. f. Hygiene, Bd. 12, 1892, p. 254.—*Burdach*. Der Nachweis v. Typhusbaz. a. Menschen. Zeitschr. f. Hygiene, Bd. 41, 1902.—*Dmochowski* u. *Janowski*. Experim. Untersuchungen über Mischinfektion bei Typhus u. über Eiterung bei Typhus. Beitr. z. path. Anat. Ziegler, Bd. 17, 1895, p. 221.—*Eberth*. Die Organismen in den Organen bei Typhus abdom. Virchows Archiv, Bd. 81, 1880, p. 58;—Neue Untersuchungen über den Baz. des Abd.-Typh. Virchows Arch., Bd. 83, 1881, p. 486;—Der Typhusbazillus u. die intestinale Infektion. v. Volkmanns Samml. klin. Vorträge, No. 226, 1883.—*Gaffky*. Aetiologie des Abdominaltyphus. Mitt. aus dem Gesundheitsamte, Berlin, 1884.—*Gaffky* und *Kolle*. Ueber Typhusschutzimpfungen. Klin. Jahrb., Bd. 14, 1905.—*Lentz*. Immunität bei Typhus. In Kolle-Wassermanns Handb. d. pathog. Mikroorg., Bd. 4, 1904, p. 894.—*E.* und *P. Levy*. Ueber das Hämolysin des Typhusbazillus. Zentralbl. f. Bakteriol., Bd. 30, 1901, p. 405.—*Neufeld*. Typhus. In Kolle-Wassermanns Handb. d. pathog. Mikroorganismen, Bd. 2, 1903, p. 204.—*R. Pfeiffer* und *Kolle*. Spezif. Immunitätsreaktion des Typhusbazillus. Zeitschr. f. Hygiene, Bd. 21, 1896, p. 203;—Experim. Untersuchungen zur Frage der Schutzimpfung des Menschen gegen Typhus abd. Deutsche med. Wochenschr., 1896, p. 735.—*Sanarelli*. Die Gifttheorie d. Abdominaltyphus. Zentralbl. f. Bakteriol., Bd. 16, 1894, p. 188.—*A. Wassermann*. Weitere Mitt. über Seitenkettenimmunität (Lymphdr., Knochenmark, Milz, Thymus als Bildungsstätten der Schutzstoffe). Berl. klin. Wochenschr., 1898, p. 209.—*Widal* et *Sigard*. Etude sur le Sérodiagnostic et sur la Réaction agglutinante chez les typhiques. Annales de l'Inst. Pasteur, T. 11, 1897, p. 353.

OTHER BACTERIA OCCASIONALLY PRODUCING PUS

Besides the above described pyogenic bacteria, there are a number of other bacteria (influenza, pneumonia bacilli) which occasionally produce suppurative inflammation. These will be mentioned when the suppurative inflammations of the different tissues are described.

The pyogenic action of the bacillus of tuberculosis, glanders, and of the actinomyces will be discussed in Part II, Chapter III, dealing with these diseases.

CHAPTER II

THE INFECTION ATRIA OF PYOGENIC BACTERIA

A WOUND of the skin or mucous membrane affords most frequently the infection atrium for pyogenic bacteria. It is incorrect, however, to regard a wound as the only infection atrium, and to suppose that one exists in each infection. Pyogenic and other bacteria may under certain conditions pass through intact skin and mucous membrane and penetrate granulation tissue.

Schimmelbusch's experiments in the production of furuncles demonstrate how bacteria may enter intact skin. The presence of the staphylococci upon the surface of the skin alone is not sufficient to produce an inflammation. A second factor is required, for the cocci must be mechanically rubbed or forced into roughened areas or fissures of the skin or into the points of exit of hairs or lanugo hair, much less frequently into the ducts of sweat glands. After the cocci have been rubbed into intact skin they may be demonstrated about the hair shaft. They multiply and pass into the hair follicle, and there produce the inflammation. From such a focus the cocci may pass into lymphatic vessels and nodes or invade the blood vessels, as the development of suppurative osteomyelitis in some distant parts following a furuncle demonstrates.

Similar experiments with a number of pyogenic and other bacteria, such as anthrax (Wasmuth) and tubercle bacilli (Cornet) have been made. All these experiments show conclusively that bacteria may be rubbed into the hair follicle, the skin being intact, and that in this way severe, or even fatal infection (animal experiments with anthrax) may be produced.

An intact mucous membrane may be penetrated by bacteria (especially by streptococci, pneumococci, less frequently by staphylococci, colon bacilli, etc). Only very virulent bacteria are able to do this, as experimental work upon the mucous membrane of the mouth cavity, pharynx, and intestines (Lexer, Bail) has shown. The bacteria pass into and multiply in the spaces produced by the continual and active migration of leucocytes through the epithelium (Stöhr's epithelial spaces) covering the lymphatic follicles (over tonsillar crypts, lingual, and pharyngeal tonsil, and Peyer's patches). The bacteria must, however, be highly virulent before they can invade the tissues, as the wandering leucocytes have bactericidal properties (*vide* p. 157).

Less virulent bacteria, as a rule, cause but little damage while they remain upon the mucous membranes, as they are prevented from penetrating them by the secretion of the cells and the movement of the cilia. The constant presence of pathogenic bacteria upon the mucous membranes of the nasal, buccal, and pharyngeal cavities, of the respiratory passages and gastrointestinal tract, and the negative results of animal experiments (Buchbinder and others), which have shown that bacteria of ordinary virulence do not penetrate normal mucous membrane, demonstrate that this is so. If, however, the mucous membranes are injured (e. g., if the protective mechanism is interfered with) the harmless bacteria will become virulent, will multiply rapidly, penetrate the mucous membranes, and produce inflammation of the deeper tissues. The protective action of the mucous membranes may be interfered with in a

number of ways: for example, by destruction or paralysis of the cilia (trachea and bronchi), by diminution of the bactericidal substances (A. Wassermann), by circulatory disturbances resulting from local or general chilling, by disturbance of circulation in intestinal strangulation, by chronic inflammation, by chemical or mechanical irritation followed by separation and exfoliation of epithelium.

The fact that wounds of the mucous membranes, especially of the nose, mouth, and pharynx, are rarely the beginning of severe inflammation, although constantly bathed, as it were, by bacteria, which when carried into operation-wounds by coughing and sneezing cause inflammation, appears to depend upon the bactericidal properties of the secretion of the mucous membranes and the leucocytes which wander through them. The rich blood supply of mucous membranes, the continual movement of the saliva, and the attenuating effects of symbiotic saprophytes are important factors in this natural resistance.

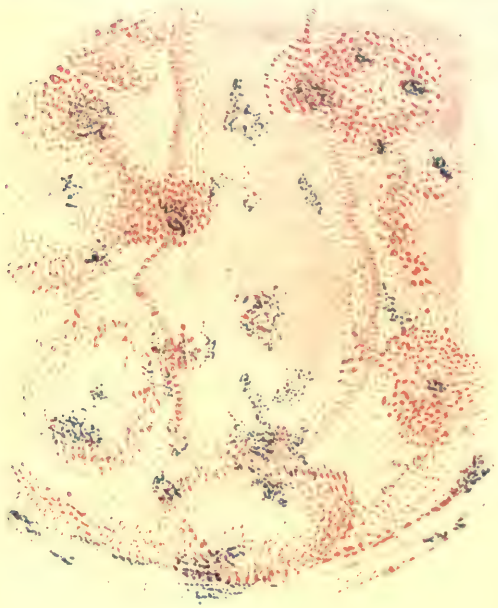


FIG. 95.—SECTION OF A TONSILLAR CRYPT OF A RABBIT WHICH DIED OF A GENERAL BACTERIAL INFECTION TWENTY-FOUR HOURS AFTER THREE DROPS OF A HIGH VIRULENT CULTURE OF STREPTOCOCCI WERE RUBBED INTO THE MUCOUS MEMBRANE COVERING THE TONSIL. The streptococci have invaded the lymphoid tissue of the tonsil, passing through Stöhr's spaces.

If mucous membranes become infected with highly virulent bacteria the inflammation may extend to the submucous tissues, producing phlegmons, may attack the lymphatic vessels and nodes or reach the blood vessels. The relations between acute angina and articular rheumatism, suppurative osteomyelitis and metastatic infection, between enteritis and general infection (*vide* Bacterium Coli Commune), are examples of the extension of infection through mucous membranes which are often quoted.

Uninjured granulation tissue is not permeable even to highly virulent bacteria and their toxins. Billroth showed that he could keep putrefactive substances and pus in contact with a large granulating wound upon a dog's back for some time without doing any harm. Noetzel has

demonstrated that granulating wounds of sheep, which are very susceptible to anthrax and tetanus, resist highly virulent cultures. The lessened susceptibility of granulation tissue to infection was known to the old surgeons, and they desired to obtain in their wounds (e. g., in plastic operations) good granulations as quickly as possible. The secretion of the granulation tissue removes the bacteria mechanically, and besides contains bactericidal substances (Afanasieff). The cells of the granulation tissue, like those of the epidermis, prevent the penetration of bacteria, but are not as resistant as the latter. They are easily torn, and if once the thin veil covering the surface of the granulation tissue is injured, the lymphatic vessels and blood vessels stand open to receive the bacteria. Therefore Noetzel was able to produce fatal anthrax or tetanus infections as soon as he injured and then infected the granulation tissue.

The experiments above cited agree with clinical experience. The yellowish, dirty membrane consisting of secretion and colonies of bacteria, which covers unhealthy granulation tissue, carries with it no added danger, for neither the bacteria nor their toxins are absorbed. If, however, the granulating wound is injured by traction upon the wound edges during the removal of adherent dressings, by the use of a caustic or sharp spoon, severe inflammation (lymphangitis, erysipelas) may develop if virulent bacteria—for example, streptococci—are contained within the granulations.

The skin affords most frequently infection atria for staphylococci and streptococci; the mucous membranes of the mouth and pharynx, nose, and accessory sinuses, the ear and respiratory passages for the streptococcus and pneumococcus; of the upper part of the gastrointestinal canal for the streptococcus and staphylococcus; of the lower part of the intestine (likewise the bile passages) for the bacterium coli commune, more rarely for the streptococcus and staphylococcus. In the urinary passage, besides the gonorrheal infections, infections with the colon bacillus, streptococcus, staphylococcus, and bacillus pyocyaneus occur; in the female genital canal, besides gonorrhea, infections with the streptococcus alone or associated with other bacteria are most frequent.

LITERATURE.—*Afanasieff.* Ueber die Bedeutung des Granulationsgewebes bei der Infektion von Wunden mit pathog. Mikroorganismen. Zieglers Beitr., Bd. 22, 1897, p. 11.—*M. Bail.* Die Schleimhaut des Magendarmtraktus als Eingangspforte pyog. Infektionen. Arch. f. klin. Chir., Bd. 62, 1900, p. 369.—*Buchbinder.* Experim. Untersuchungen am lebenden Tier- u. Menschendarm. Deutsche Zeitschr. f. Chir., Bd. 55, 1900, p. 458.—*Helmberger* und *Martina.* Experim. Untersuchungen über die Durchgängigkeit des Darmes f. Bakt. Deutsche Zeitschr. f. Chir., Bd. 74, 1904, p. 527.—*Jürgelünas.* Ueber die Durchgängigkeit des Granulationsgewebes f. pathog. Mikroorganismen. Zieglers Beitr., Bd. 29, 1901.—*Lexer.* Die Schleimhaut des Rachens als Eingangspforte pyog. Infektionen. Arch. f. klin. Chir., Bd. 54, 1897, p. 736.—*Nötzel.*

Ueber die Infektion granulierender Wunden. Chir.-Kongr. Verhandl., 1897, II, p. 272 and Arch. f. klin. Chir., Bd. 55, p. 543.—*Schimmelbusch.* Ueber d. Ursachend. Furunkel. Arch. f. Ohrenheilkunde, 1889, Bd. 27, p. 252.—*Wasmuth.* Ueber die Durchgängigkeit der Haut für Mikroben. Zentralbl. f. Bakteriol., Bd. 12, 1892, p. 824.—*A. Wassermann.* Infektion und Autoinfektion. Deutsche med. Wochenschr., 1902, p. 117.

CHAPTER III

PYOGENIC INFECTIONS AND THEIR TREATMENT

The invasion of the tissues follows closely the infection of the wound with pyogenic bacteria. Pyogenic, like all other bacteria, require a certain but short period, the so-called incubation period, before they actively invade the tissues. During this time they multiply, their virulence increases by growth upon the good culture media furnished by the tissues, and their own power is increased and the development of bactericidal substances in the wound secretion is retarded.

After the invasion of a wound or of the uninjured mucous membrane or skin the bactericidal substances and protective mechanism are able to cope for the time being with the bacteria. The beginning struggle between the bacteria and the tissues with its victories and defeats pursues an acute, rarely a chronic course, which usually but not always ends in suppuration. If the bacteria are weak and few in number, they succumb to the bactericidal substances in the tissue fluids and the inflammation is mild, ending without pus formation. No pus may be formed, but extensive necrosis may result when the bactericidal substances are too weak to resist the numerous and highly virulent bacteria. Between the mild and virulent infections there is a variety which ends in pus formation. When the struggle between the bacteria and tissues is about even, pus is formed. The invasion of new tissue indicates a victory for the bacteria, while the subsidence of the inflammation and the encapsulation of the pus indicate that the bactericidal substances of the tissues have prevailed. An acute suppurative inflammation is therefore characteristic of pyogenic infections, although other forms of inflammation, excepting the putrefactive forms, may be present (*vide* Inflammation).

If bacteria and their toxins are absorbed the struggle is transferred to the whole body and becomes general. More forces are then at the disposal of the organism to combat the infection, which, however, are often denied when the infection is virulent or the general condition poor. A lymphogenous or hæmatogenous infection, depending upon

whether it is carried by the lymph or blood stream, is distinguished from an ectogenous infection in which the bacteria gain access from the outer world, and an endogenous infection in which the bacteria have lain dormant for some time upon the mucous membranes (e. g., of the intestine or bladder) or in an old encapsulated focus.

The action of pyogenic bacteria as of all pathogenic micro-organisms depends upon their ability to produce toxins during their growth in the tissue, and in their death and solution to liberate their protoplasmic toxins.

The methods which should be employed in the treatment of pyogenic infections depend upon the cause of the infection, the character of the inflammation, and the complications and sequelæ which most frequently follow. The treatment should in no way interfere with the inflammatory processes which combat the infection, and should therefore not be antiphlogistic. The tissues involved should be aided in their struggle against the bacteria, and the inflamed part should be kept quiet by an immobilizing dressing and protected from external injury, and the inflamed area should be incised to permit of the escape of the bacteria and their toxins, in this way lessening their action. If efficient treatment is instituted early, the tissues will be aided in their struggle against the bacteria and will overcome the infection with but little destruction of tissue.

Absolute Rest, Obtained by Immobilizing Dressings, Elevation, etc.— In the beginning of any inflammation absolute rest, obtained by the use of an immobilizing dressing which exerts no pressure is of great importance. Frequently it alone will control mild inflammation and cause it to subside. Suspension or elevation of an inflamed extremity is useful, as it favors the venous return and prevents stasis in the inflamed area, which hastens the destruction of tissue.

Frequently well-fitting bandages combined with elevation will alleviate the pain resulting from the inflammation. Its action is increased if dressings covered with an ointment (five to ten per cent zinc oxide or boric vaseline) are used, as they impart to the tense, inflamed skin a pleasant, cool sensation. The ointments have this advantage, that they do not macerate the skin as moist dressings do, and besides they may be allowed to remain in position a long time (a number of days), and then there is no occasion to disturb the immobilizing dressing.

Cold in the form of an ice bag has been used extensively because it controls pain. Applied for a short time in superficial inflammation, it lessens the hyperæmia by contracting the blood vessels and delays the inflammatory process. If used for a longer time there is an increased tendency to stasis, and necrosis may occur. The ice bag is to be recommended for the control of pain only when the deeper tissues are inflamed,

or in peritonitis when one hopes to delay the inflammatory process and to localize or encapsulate the inflammation.

Hot, Moist Compresses.—Moist compresses (three per cent acetate of aluminum, or boric acid solution, ninety-five per cent alcohol according to Salzwedel, with or without a rubber or gutta percha covering) control the pain, but often injure the skin. In mild inflammations, the moist compress, with the help of the hyperæmia which it induces, often causes the inflammation to subside without suppuration. In severe inflammations, on the other hand, moist compresses produce an increased exudation, the result of the greater hyperæmia. The increased pressure in the inflamed area then drives the bacteria and their toxins into the surrounding tissues, and the tissues become necrotic and are liquefied by the ferments in the pus. For this reason moist compresses and still more hot poultices (cataplasms) hasten and increase the destruction of tissues and the formation of pus. For example, a subcutaneous felon treated for eight days by poultices extends to the tendon and bone (*vide* Fig. 96). The author has observed a complete sequestration of the glandular substance of the breast in a mastitis which was treated for two weeks with poultices. Moist compresses may be used in large, deep-lying, indurated areas to " ripen the abscess " so that the knife may be used.

Fig. 96.—A Felon of the Index Finger Associated with Necrosis of the Flexor Tendon and Destruction of the Joints Following an Injury of the Skin. Result of two weeks' treatment with poultices.

It is a gross error, but one which is frequently made, to use an ice bag or moist compress upon the extremities in place of the immobilizing dressing.

Incision of Abscess and Evacuation of Pus.—As soon as pus is demonstrated by the sensation of fluctuation, or suspected because of the location of or increase in size of the swelling (e. g., in submaxillary region in periostitis of the mandible, inflammation of tendon sheath of the hand), or because of the clinical symptoms alone (e. g., in the body cavities), an incision should be made and the pus allowed to escape. The

incision should be large enough to permit of the escape of the pus, and to reduce the tension of the tissues. In making the incision the tissues should be divided layer by layer, as in this way the important anatomical structures may be most easily avoided. Puncture of an abscess alone or drainage through a small opening is always insufficient, as is also the expectant treatment, in which the pus is allowed to break through the skin. Deep suppurating foci in bone, in the brain and skull, and in the thorax should be rendered accessible by special operations.

Anæsthesia and ischæmia of the part involved (excepting in case of accompanying lymphangitis) are indispensable in opening large phlegmons and deep-lying abscesses if the operator wishes to find all the recesses, and to avoid all the important nerves, ligaments, etc.

The method of local anæsthesia suggested by Oberst is best suited for opening abscesses of the finger tips. In this method a piece of rubber tubing is applied about the base of the proximal phalanx, and then the cocain is injected into healthy tissues along the course of the sensory nerves (*vide* Local Anæsthesia). Injections should not be made into the inflamed tissue, as in Schleich's method, as the bacteria may be forced into the surrounding and deeper tissues.

Small abscesses, furuncles, and small carbuncles may be incised after the area has been frozen with ethyl chloride. The tissues are frozen more rapidly with this than with the ether spray.

Avoidance of Mechanical and Chemical Irritation.—The incised wound should not be irritated mechanically or by chemicals. The infected tissues are injured and their resistance reduced by pressure, sponging, irrigation, washing out of pus, or the curetting of necrotic tissue, by separating tissues with fingers or hooks, by probing the wound—in short, by all rough procedures. As a consequence of such manipulations, bacteria or their toxins may be forced into the blood, and the lymph vessels or thrombi may be separated. Then, instead of the fever falling after the incision and the inflammation subsiding, it extends, accompanied by chills and fever, or lymphangitis or erysipelas develops about the edges of the wound, or dangerous lung embolism, metastatic inflammation, or general infection results.

Chemical irritation leads to a superficial necrosis of the surfaces of the wound. Pure carbolic acid, as recommended by von Bruns to be applied for one minute, has certainly an immediate sterilizing action, but the eschar which it forms prevents the discharge of the exudate loaded with bacteria and toxins, and these are driven into new paths. The ordinary antiseptic solutions do not injure the tissues if they act for a short time, but they likewise have no effect upon the bacteria in them. They kill only those bacteria upon the surface of the wound, and must act for some time even to produce this result. The tissues

are injured, their resistance is reduced, or they become necrotic (*vide* Carbolic Necrosis) when antiseptic solutions are used. Paste-like agents (Schleich's wound preparations, glutol, glutol-serum) and powders (iodoform, subnitrate of bismuth) should not be used, as they form a crust which prevents the discharge of the secretion.

Tampon and Tubular Drainage.—A freshly incised wound should be loosely tamponed with iodoform gauze, which should provide drainage for all the recesses and cavities. Dry gauze placed in a wound acts in two ways, it controls the hæmorrhage and removes by its capillarity infectious materials, and therefore retards the post-operative absorption. The exudate and blood lying in the recesses of the wound and seeping from the tissue are carried by the capillarity of the gauze into the dressings. Moist gauze saturated with antiseptic solution has no such action. As moist gauze dries it acquires capillarity, and then resembles more closely in its action iodoform gauze. The bactericidal action of gauze saturated with antiseptics when compared to the action of dry gauze is of secondary importance.

In deep wounds with recesses the tampon should be combined with tubular drainage. A large rubber drain should be inserted into the deepest part of the wound, and this should be surrounded by iodoform gauze. The moist tampon, evaporation from which is prevented by a rubber covering, has no capillarity whatever. This alone is enough to condemn this dressing, but there is still another objection, as bacteria multiply rapidly within the gauze in spite of the antiseptics it contains, upon the skin, and within the wound.

The tampon should not be removed for twenty-four hours, for usually the surface of the wound is then freshly injured and absorption is favored. The tampon should be changed at the end of forty-eight hours, as it then no longer conducts away the wound secretion, but prevents the discharge of that which is newly formed.

Care should be exercised in changing tampons and dressings to avoid injuring the wound surfaces and providing new infection atria. The tampon, if adherent, should be moistened with a three per cent hydrogen peroxide solution, and with the development of the foam the gauze becomes loosened, and can be removed without the least hæmorrhage.

The greatest care should be exercised at each dressing. Thick pus formed in large amounts may be made to flow from deep wounds by changing the position of the patient, or may be removed by careful irrigation with sterile water (without pressure), or may be gently wiped away. Pus upon the surface of a wound does less injury, even when allowed to remain, than the rough attempts at removal, which may be followed by lymphangitis, erysipelas, fever, etc. The edges of the wound should be covered with an ointment to prevent the dressings from

becoming adherent. Sterile gauze or mull, impregnated with some substitute for iodoform (dermatol, xerol, Crede's silver preparation, etc.), may be used instead of iodoform gauze if there is a tendency to eczema.

The incised wound must be kept open by a superficial tampon, or drained as long as the discharge of pus is profuse. The conditions in the wound change after a few days, and the line of treatment must be changed to meet conditions. The tampon takes up the blood, the tissue fluids, and the remaining pus from the surfaces of freshly incised suppurating wounds. After a few days, when the granulation tissue develops and the necrotic tissue becomes separated, the wound discharges a thick creamy pus which iodoform gauze will not drain. When the pus becomes thick and creamy a tubular drain should be inserted into the deep parts of the wound.

In such conditions ointment dressings or moist compresses without the covering of rubber tissue are indicated. They produce a mild irritation resulting in hyperæmia, which hastens the separation of necrotic particles, thins the pus by increasing the exudate, and cleanses the granulating surfaces. If the pus or the membrane covering the granulation tissue is removed mechanically, there is always the danger of new infection, for the granulating surface absorbs as soon as it is injured.

Mercury ointments (unguentum hydrargyri cinereum, hydrarg. oxyd. rubr., four to eight per cent with vaseline; mild, ten per cent zinc salve) are best suited for these cases. Three per cent acetate of aluminum, two per cent boric acid solution and alcohol are used for moist dressings, and if the evaporation is not prevented by the use of rubber tissue, the gauze as it dries acquires a strong capillary action (*vide* p. 29).

The treatment of healthy granulating incised wounds is the same as that of a fresh wound healing by granulation tissue. Immobilizing of the inflamed part and rest in bed should be maintained until the tense reddened skin becomes shrunken and pale, the temperature falls, and the coated granulations become clean. A dressing poorly applied permits of muscular action which injures the inflamed tissues. Bandages which exert pressure and constrict the parts produce circulatory disturbances, retard the escape of wound secretion, and favor the extension of the inflammation. One should therefore learn to properly apply a bandage in acute inflammations. Von Volkmann's wooden splint or a simple papier maché splint, well padded, may be used to immobilize the extremities.

Prophylaxis.—Prophylaxis is an important factor in the treatment of all acute suppurative and putrefactive infections. All patients with erysipelas, phlegmons, suppurative osteomyelitis, metastatic infections, etc., should be placed in an isolation ward, which every well regulated

hospital should have at its command. The staff of this isolation ward should not have access to the operating room or any other ward. While dressing patients the physician should wear rubber gloves to protect his hands, and remove the saturated dressings with large dressing forceps, for it is possible to transfer bacteria into other wounds when the fingers have been in contact with pus, even when the hands have been thoroughly sterilized. (For the same reason a surgeon should always wear rubber gloves when he performs an autopsy.) It scarcely need be emphasized that the gown should be changed after the dressings have been completed.

Concerning the use of Bier's passive hyperæmia in acute infections, see Chapter VII, page 310.

CHAPTER IV

THE PYOGENIC INFECTIONS OF DIFFERENT TISSUES

(a) THE PYOGENIC INFECTIONS OF THE SKIN AND SUBCUTANEOUS TISSUES

FURUNCLE

If the pyogenic micro-organisms which inhabit the epidermis, or are transferred to it from neighboring suppurating foci or by infected fingers are forced into the pores of the skin by rubbing, a circumscribed inflammation develops (Garré, Bockhart, Schimmelbusch). The bacteria gain access much more easily to the points at which the hairs pierce the skin than to the tortuous ducts of sweat glands, and so, as a rule, the cocci develop along the shaft of the hair or lanugo hair and reach the hair follicle and its sebaceous gland (Schimmelbusch, *vide* p. 193).

They produce a circumscribed, acute, suppurative inflammation, the central point of which is the hair which provided the infection atrium. The hyperæmia and exudation into the tissues produces within a few days a round, somewhat elevated, deep red, hard nodule, which develops, accompanied by an increasing burning pain, sometimes to the size of a pea, sometimes to that of a cherry. The formation of pus causes an elevation of the epidermis in the center of the red area, and a yellow pustule develops at the base of the hair involved. After the pus is discharged the small inflammatory focus heals. In large furuncles there is a necrosis of tissue. The hair follicle and sebaceous gland from which the bacteria penetrate into the deeper surrounding tissues are destroyed. The larger the inflammatory focus becomes, the greater the amount of tissue surrounding it destroyed. The necrotic tissue which is

separated by the pus has the form of a cone, the apex of which extends into the subcutaneous tissues. Left alone, the necrotic tissue or " core " is extruded or liquefied after one or two weeks. The space left in the tissues after the removal of the " core " is filled in by granulation tissue and healing occurs, while the reddened area disappears within another week.

Bacteria Found in Furuncles.—The type of inflammation above described is the furuncle or boil; smaller and larger foci may be regarded as varieties of the same. The most frequent causes of the furuncle are different varieties of the staphylococcus, particularly the aureus and albus, yet streptococci may be found which, in spite of the stubborn course of the infections, produce at most a pustule, but no extensive necrosis. The furuncle is therefore most often a *staphylomycosis circumscripta cutis* (Kocher).

Etiological Factors: Mechanical Irritation, Uncleanliness, Diabetes, etc.—Furuncles develop most frequently upon parts of the body which are exposed to mechanical irritation (rubbing by clothing) and which are often unclean (nates, inner surface of the thigh, axillary fossa, neck). They develop also in inflamed areas (resulting from eczema, prurigo, urticaria, scabies, and vermin of all sorts) which are scratched. They may develop simultaneously in different parts, or stubborn reinfections may develop in a patient whose resistance to pyogenic infections has been reduced by some disease (diabetes, cachexia, marasmus). In those parts in which comedones are frequent (face and back), furuncles are often intermingled with the harmless acne pustules or develop from them. If a furuncle develops after an insect sting or bite, it is hard to say whether the infection was transferred by the insect or by the fingers when scratching. A typical furuncle develops after a prick with a needle infected with staphylococci. In the palms of the hands and the soles of the feet furuncles rarely develop, as there are no hair follicles. If they do develop here the sweat glands or slight injuries afford the infection atrium.

Complications: Lymphangitis, Lymphadenitis, General Infections, etc.—As a rule, a progressive inflammation does not develop from a single furuncle. The inflammation remains circumscribed, and after the extrusion of the necrotic center (" core ") there is a tendency to heal. Healing occurs spontaneously, as a rule, in from two to three weeks. In spite of this every furuncle carries with it a number of dangers. In the first place, the pus discharged by an open boil, which is not dressed, may be carried by the clothing, fingers, or poultices to surrounding skin. Other furuncles are produced in this way, which in diabetics or in weak nursing children may extend over the entire body (furunculosis). In the second place the bacteria may enter the lymph or blood vessels. Fre-

quently infectious substances are absorbed from a single boil, and there is an elevation of temperature accompanied by the other symptoms of fever. It is easily understood why lymphangitis and lymphadenitis frequently develop; this happens frequently with furuncles of the hands and feet as the movements of the clothing favor the entrance of the cocci into the lymphatic spaces. Blood infections may develop from furuncles. In these cases the bacteria either invade the capillaries and small veins, infected thrombi are loosened spontaneously or by rough manipulations (pinching of the furuncle, curettage after incision), or a large vein (facial, saphenous) adjacent to the furuncle becomes inflamed. Any furuncle may become dangerous by causing general pyogenic infections (osteomyelitis, metastatic suppuration of joints, viscera, etc.), and this should be kept constantly in mind during the treatment.

Varieties of Furuncles.—*Carbuncles.*—Carbuncles, the worst form of furuncle, which develop frequently in old age, in children, and young adults, are often associated with these dangers. A carbuncle develops as the result of simultaneous infection of adjacent hair follicles, or when hair follicles adjacent to a furuncle become infected by the pus discharged from it. It is characterized by considerable pain and swelling, rapid growth, extensive destruction of tissue, high fever, and severe general symptoms. The center of the bluish red, infiltrated area, which rises about two fingers' breadth above the healthy surrounding skin and gradually slopes into it, contains pustules and necrotic tissue, or is covered by a hæmorrhagic, purulent crust or gangrenous skin. If the inflammation extends or new furuncles develop as the result of improper (poultices) or no treatment, the carbuncle rapidly increases in size and may become in from one to two weeks as large as a hand or a small plate (Fig. 97). The inflammation then extends to the fascia covering the muscles; the tissues become infiltrated and riddled with small purulent and necrotic foci. The tissues in the center of the carbuncle are destroyed, and frequently the necrosis extends so deep that pieces of the fascia are extruded. Thrombophlebitis of the subcutaneous veins may develop, which favors the extension of the inflammation to neighboring tissues; in carbuncle of the upper lip the facial, the angular, superior ophthalmic veins may become involved, and process may extend to meninges through the cavernous sinus, or give rise to a general pyogenic infection.

The lymphangitis associated with carbuncles of the extremities is easily seen. Often the neighboring lymph nodes suppurate. In weak individuals and diabetics a carbuncle may produce a chronic febrile condition which may lead to cachexia and death, without meningitis or metastatic foci developing.

The development of a carbuncle depends partly upon the condition of the patient—e. g., diabetes—and partly upon the virulence of the bacteria.

Besides staphylococci, all possible forms of pyogenic bacteria, particularly the streptococci, may be found in a carbuncle. According to Kocher, the streptococci alone may produce this variety of inflammation. Frequently they develop from furuncles which have been pinched or stuck by the patients themselves, or after the use of moist dressings or poultices, which increase and hasten suppuration and necrosis, macerate the healthy skin surrounding the furuncle, and inoculate it with pus which is not absorbed by the dressing.

FIG. 97.—LARGE CARBUNCLE OF THE NECK DEVELOPING FROM A FURUNCLE IN TWO WEEKS UNDER TREATMENT WITH POULTICES. The patient is a powerful nondiabetic man.

Folliculitis Barbæ.—Those inflammations which are limited to the hair follicle and its sebaceous gland (folliculitis) are harmless forms of furuncles. In hairy parts small pustules with little infiltration develop. If the hair is removed, and with it the thick pus surrounding its root, the inflammation subsides in a few days. If in the beard, the infection extends from one hair follicle to another, the stubborn sycosis or folliculitis barbæ develops, which demands the irksome removal of all the hairs, the opening of all newly formed pustules, and the use of salve dressings.

Other forms of follicular inflammation develop upon parts of the face and back where comedones occur. The duct of the sebaceous gland, occluded by dirt, dilates the opening at which the lanugo hair pierces the skin to such an extent that an infection atrium is provided.

Frequently the expression of the comedo with fingers infected by pus from an adjacent pustule, or which are dirty, is the exciting cause. A blackhead occupies the center of the small red furuncle, which is never larger than a pea. This form of furuncle develops frequently in

unclean individuals in whom there is a tendency to comedones, particularly at the age of puberty. It has been called acne punctata, or if a pustule forms, acne pustulosa. An inflammation of the cilia and their sebaceous glands (Meibomian glands) is called a hordeolum.

Prognosis.—Healing occurs within a few days after rupture or removal of the pustule with forceps or by incision. The inflammation persists if the inflamed area is repeatedly pinched by the patient, or the pus carried into surrounding tissues and new furuncles develop.

Diagnosis.—The diagnosis of the carbuncle offers the most difficulties, for it may be confused with the carbuncle produced by anthrax, for the latter may develop a pustule when there is secondary infection. The microscopic demonstration of the anthrax bacillus settles the diagnosis.

Treatment.—The treatment of a furuncle should promote rapid healing and prevent the development of other furuncles. If the furuncle is so situated that it is exposed to friction, it must be protected by a gauze dressing, which is retained in position by adhesive plaster. In this way it is protected from irritation from without, and the pus discharged from it is not carried to adjacent skin. If the infiltration increases rapidly, although the furuncle has discharged spontaneously or has been opened (removal of the hair, opening with tissue forceps), it should be incised.

FIG. 98.—THE SAME CASE EIGHT DAYS AFTER OPERATION. Healthy granulation tissue has developed and the four flaps which were dissected free to their bases are again united.

After sterilization of the skin an incision is made under ethyl chloride anæsthesia, as wide and deep as the infiltration extends. Some layers of iodoform gauze are placed in the wound, and when these are removed twenty-four hours later the completely separated core will be found attached to it. Large furuncles heal under this dry and sparing treatment in one week without any sequelæ. A small salve dressing is applied after the core is extruded. At each dressing the surrounding area should be sterilized with alcohol.

Moist Dressings and Poultices.—Moist dressings and poultices hasten the softening of the furuncle, but do not hasten repair. The infiltration increases, the necrotic core becomes larger, while the pus under the dressing infects healthy surrounding skin, which is macerated and made more susceptible to infection. As a rule a number of other furuncles develop when moist dressings are used. Moist antiseptic dressings have as little effect upon the bacteria in the surrounding skin as they do upon those in the furuncle.

In the large furuncles and carbuncles a simple incision is never sufficient. A crucial incision must be made under general anæsthesia. The flaps formed in this way are held apart with retractors, and an incision is made so that the infiltrated tissue is separated from the healthy, and the entire inflammatory focus is exposed. The corners of the flaps are either necrotic, or will become so, so it is best to remove them at once. The wound is kept wide open to permit of the discharge of pus and hasten the separation of any remaining necrotic tissue. Without doing any damage, attempts are made to remove whatever necrotic tissue may be easily separated; the larger vessels are then ligated and the wound tamponed with iodoform gauze, which should remain in position, as a rule, two days, until loosened spontaneously. When the gauze is removed granulations are already present. [Moist dressings of boric acid solution and gauze are believed by some surgeons to be of great value, and if the field of the lesion is sterilized by alcohol at each change of the dressing, there is little danger of spreading the infection. They afford much comfort to the patient.]

Fig. 99.—The Same Case Four Weeks After Operation.

Granulation tissue fills in the defect resulting from a large carbuncle in one week, and becomes covered with epithelium in from one to two weeks later. The scar then contracts (Figs. 97 and 99). The inflammation does not subside, neither does the fever fall, when small, insufficient incisions are made.

When thrombophlebitis of one of the larger veins develops, the vein

should be opened, after proximal ligation, and the suppurating thrombus removed.

Frequently the incision is postponed too long. In furuncles or carbuncles of the face it is dangerous to delay, for here the large number of lymphatics and the frequent occurrence of thrombophlebitis of the facial vein may lead to fatal results through general infection or meningitis.

General infection is prevented by an incision correctly made and proper after-treatment, although some believe that incision favors general infection. General infection may be easily produced by curetting out the pus, squeezing and irrigating the furuncle, and by any other form of mechanical irritation (*vide* General Rules for Treatment, p. 199).

Successful treatment is more difficult in those cases in which a number of furuncles develop upon different parts of the body (furunculosis). In these cases the operative treatment must be combined with measures which prevent the development of virulent staphylococci. Daily warm baths (with salt water, green soap, etc.) should be taken, the clothing should be changed after each bath, new dressings applied to open furuncles and those which have developed opened. Ointments and plasters favor the infection of adjacent areas. If the furunculosis is limited to one part of the body, one application of a five per cent formalin compress, which may be allowed to remain for some hours, may be of great value.

It is understood that diabetics should receive appropriate internal treatment. In severe cases heart stimulants can rarely be dispensed with.

THE SUBCUTANEOUS ABSCESS

Any inflammation developing in the deeper tissues may lead to an accumulation of pus in the cutis and subcutaneous tissues. Small abscesses following wounds are rarely limited to the skin; they extend to or develop in the subcutaneous tissues. Abscesses due to the imperfect opening of a furuncle spread in this tissue, and circumscribed suppuration occurring with erysipelas, lymphangitis, and subcutaneous phlegmon develops here. All deep-lying suppurating foci extending outward form collections of pus in the meshes of the loose connective tissue as soon as the subcutaneous tissue is reached, as, for example, after rupture of suppurating foci of the body cavities, joints, bones, muscles, and all deep-lying abscesses.

If subcutaneous hæmatomas following injuries become infected from an excoriation or wound, an abscess is formed. Lymphogenous and hæmatogenous infections of hæmatomas as well as the ectogenous may

occur. Metastatic abscesses in the subcutaneous tissues in all parts of the body, as well as metastatic abscesses of muscles, organs, and joints, may occur in general pyogenic infections, particularly after staphylo-

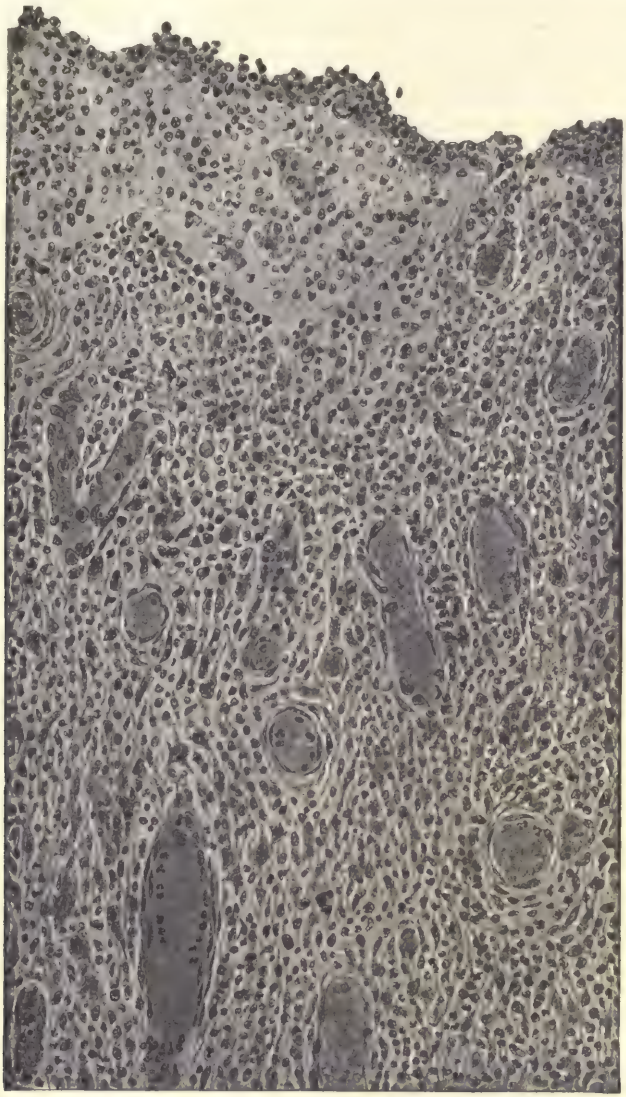


FIG. 100.—THE INTERNAL LAYER OF AN ABSCESS MEMBRANE COMPOSED OF GRANULATION TISSUE. Newly formed capillaries lie within a cellular tissue composed of fibroblasts, leucocytes, lymphocytes, and fine fibrillæ.

coccic infections. In rare cases after typhoid fever, the typhoid bacilli are the cause of these general infections.

The subcutaneous abscess is characterized by swelling, redness, and tension of the skin, pain, local elevation of temperature, and fluctuation in the center of a hard, infiltrated area.

It is accompanied by a moderate, often continuous fever. The skin covering the center of the abscess becomes thin and bluish sooner or later and opens spontaneously if not incised. After the discharge of the pus the inflammation may subside and the abscess heal. The longer the pus remains in the tissues, the more extensive the granulation tissue which walls it off. This abscess membrane, composed externally of connective tissue bundles, internally of granulation tissue, harbors the pyogenic bacteria, which in large cavities maintain a discharge of pus for a long time from the point of rupture (fistula). When the bacteria and necrotic tissue have been discharged in this secretion, the opposing walls of the abscess grow together and healing occurs.

As a rule, abscesses pursue a mild clinical course. If, however, the encapsulating membrane is ruptured by trauma, massage, or movements, the inflammation may extend and invade lymphatics or blood vessels.

Abscesses heal rapidly when incised and subsequently tamponed or drained. They should never, however, be regarded too lightly.

THE SUBCUTANEOUS PHLEGMON

A progressive inflammation, which is most often superficial, of the subcutaneous or of the loose connective tissues filling anatomical spaces (e. g., of intermuscular connective tissue surrounding the œsophagus, of the mediastinum), is called a phlegmon.

If the inflammation is caused by pyogenic bacteria, pus is formed; if by putrefactive bacteria, there is a tendency to gas formation and gangrene (*vide* Putrid Inflammation).

A phlegmon of the finger or toe is called a panaritium or felon. Felons are classified as subepidermal, subcutaneous, synovial, articular, and osteal, depending upon the tissues involved. Phlegmon of the periosteum is synonymous with suppurative periostitis, phlegmon of bone marrow with suppurative osteomyelitis.

A subcutaneous phlegmon may follow the pyogenic infection of a wound; the infection may occur through the blood stream in metastatic inflammation, or it may extend from deeper tissues. A subcutaneous phlegmon may extend downward and involve the fascia between the muscles, where an injury has prepared the way for the extension of the inflammation.

Phlegmons present different clinical pictures, depending upon whether the serous or suppurative type of inflammation predominates and the extent of the necrosis. One differentiates a serous, a suppurative, and

necrotic phlegmon, but the lines of separation are not strictly drawn, because transitions are frequent.

The essential requirement in the development of a phlegmon is a bacterial invasion of the subcutaneous tissue. The cutis and the fascia which limit the inflammation are only involved secondarily. The bacteria most frequently found in phlegmons are the staphylococci or streptococci. Often both are found; frequently they are associated with other pyogenic bacteria.

Streptococci produce the severest forms of phlegmon, acting alone or combined with other bacteria. They are particularly virulent when they come from a streptococcic infection in another individual. Injuries during post-mortem examination of fresh cadavers with suppurative peritonitis, meningitis, or general infections are frequently the cause of the most malignant forms of phlegmon and wound infection.

The painful swelling and redness of the skin, associated with an inflammatory œdema of the surrounding tissues and severe general symptoms, develop rapidly. The fever, which, as a rule, begins with a chill, rises rapidly and is in the beginning continuous. Later, when the general symptoms become pronounced, there is a decided morning remission.

The neighboring lymph glands become swollen and painful early. Suppurative lymphadenitis, superficial lymphangitis, and thrombophlebitis of the subcutaneous veins are often the results, but also frequently the cause of the phlegmon. Bacteria are frequently found in the blood when the bacteriological examination is correctly made (*vide* Blood Examination in General Infections).

A circumscribed phlegmon is differentiated from a diffuse phlegmon, depending upon the local course of the infection. The former subsides even after an acute onset, after a moderate extension. The virulence of the bacteria and their toxins is reduced by the resistance of the tissues and their juices. The phlegmon may run a subacute, often a chronic course, and one or several abscesses may form if a wall of granulation tissue develops which prevents the extension of the inflammation. Trauma or rough handling (massage) may easily excite inflammation again.

The progressive phlegmon does not subside unless it is incised. It pursues an acute, even violent course, and often the subcutaneous tissue of a whole extremity or part of the trunk is involved, or the infection passes to the intermuscular connective tissue and spreads incessantly.

These differences in clinical course depend partly upon the resistance of the body and the tissues involved. The diffuse phlegmon develops much more frequently in the sick and weak patient (diabetes, marasmus) than in the healthy and strong, and also in tissues which have been damaged in severe injuries.

Phlegmons, especially those which have been operated upon, fre-

quently subside and then develop again. After incision the phlegmon frequently ceases to spread. Only the severest forms cannot be controlled in this way. When incised the cutis and subcutaneous tissue are indurated, and large quantities of serous exudate are discharged from the opened subcutaneous tissue. Only a few small pockets of pus are found scattered throughout the tissue.

The clinical course of the suppurative phlegmon is less malignant when treated properly. In the beginning the serous exudate is most marked, but pus may develop as early as the second day. If incised and the pus is allowed to escape there is but little necrosis, and it is limited to the subcutaneous tissues; the phlegmon becomes limited, and the inflammatory swelling subsides. If the incision is delayed or is not large enough, if the phlegmon is treated with poultices and ointments, the inflammatory process destroys the fascia, extends to the spaces of the soft tissue, or even to the bones and joints, and gives rise not infrequently to severe general symptoms.

Only extensive incision of the phlegmon will prevent the extension of the inflammatory process and the general infection. The incision should be made—under general anæsthesia and artificial ischæmia—into the subcutaneous tissue, and should open the pockets between the muscles.

Counter-openings should be made to provide for the discharge of pus from the large pockets under the separated skin. The fever falls rapidly after incisions which have been properly made. The temperature may rise again if pus accumulates in the recesses of the wound, if the inflammation extends, or if the wound is irritated during a change of dressings (*vide* Fundamental Rules for Treatment, p. 199).

The bacteria disappear from the blood, if the inflammatory processes subside, in from one to two days and the leucocytosis disappears.

The first danger of a phlegmon is that the inflammation may extend to important organs. A thrombophlebitis of a subcutaneous vein may develop and the inflammation extend to the deeper tissues in this way. Meningitis may follow a phlegmon of the face or scalp, mediastinitis and œdema of the glottis may follow a phlegmon of the neck. The second danger is that an infected thrombus may be loosened and that fatal lung embolism, lung abscess, or metastatic infection may result. The third and greatest danger is general infection with bacteria and their toxins, overwhelming the body, which can no longer offer resistance. If the phlegmon is not controlled by incision a fatal general infection may develop unless an amputation is possible and is performed at the proper time (von Bergmann, Heinr. Wolff). Severe symptoms, extension of the inflammation, persistence of demonstrable blood infection, decline of the patient in spite of extensive incisions and free discharge of the pus are indications for this radical procedure.

The rest of the treatment is the same as that employed for acute, suppurative inflammation.

Rest in bed until the temperature reaches normal is necessary. The general treatment should sustain the heart, which becomes weak in long-continuing fever, and strengthening nourishment should be given.

Large granulating surfaces, which form in gaping wounds and after the necrosis of large areas of skin, must be skin grafted when in good enough condition. Scars which produce contractures of the fingers or joints may be frequently stretched. Only keloid thickenings should be excised, and then an attempt should be made to unite the edges of the defect or it may be skin grafted.

ERYSIPELAS

Erysipelas, derived from ἐρυθρός, meaning red, and πέλλα, skin, was recognized by the ancients as the most frequent wound infection. It runs its course with an acute, progressive reddening of the skin or mucous membrane, accompanied by fever. It develops much less frequently since the introduction of antisepsis and asepsis.

Bacteriology of Erysipelas.—Fehleisen (1881) microscopically, by cultivation (1883), demonstrated that a streptococcus was the cause of erysipelas. For a long time it was called the streptococcus of erysipelas, and distinguished from the streptococci derived from pus, mucous membranes, and other sources. Clinical experience, and especially Petruschky's experiments and investigations, have shown that there is no streptococcus which produces erysipelas alone and is specific for it (*vide* Streptococcus Pyogenes).

This explains why pus of all kinds, if it gains access to a wound of the finger during an operation or post-mortem examination, may produce erysipelas.

Often erysipelas, without a new infection, follows the incision of a phlegmon or the aspiration of an empyema of a joint, for the pus contains streptococci which enter the spaces of the cutis which have been opened. The relation between erysipelas and puerperal sepsis is explained in this way. The child of such a patient develops an erysipelas of the navel, and the physician who confined the patient an erysipelas of an injured finger. The reverse may also happen; the lying-in woman develops puerperal fever after being confined by a midwife who has been in contact with erysipelas, or who has recently been sick with the same.

Explanation of Epidemics of Erysipelas in Hospitals.—It is easily understood why epidemics of erysipelas developed in surgical hospitals in preantiseptic times, when one considers the extensive distribution and the resistance of streptococci.

The surgeon and his assistants carried the streptococci from wound to wound; their hands, the instruments, operating table, linen, etc., becoming infected with virulent streptococci when they incised an abscess. The view that the desquamated skin contained streptococci and that infection occurred through the air was disproven by Respinger and others. Erysipelas is not contagious—that is, there is no direct transference of the disease.

The development of the disease depends first upon a strain of streptococci,[1] which is virulent enough to overcome the bactericidal properties of the tissues or becomes so after remaining in the tissues for a short time; and secondly, upon the involvement of the lymphatic spaces of the cutis or mucous membrane, after which, in from one to two days (according to Fehleisen after fifteen, at most sixty-one hours, after inoculation of man), the first local symptoms develop.

The invasion of the skin or mucous membrane may occur in three ways. We distinguish between an *ectogenous*, a *lymphogenous*, and a *hæmatogenous erysipelas.*

The *ectogenous* form is by far the most frequent. There is always an epithelial defect in the skin or mucous membrane which provides the infection atrium. Rhagades, ulcers, insignificant changes of the skin and mucous membrane, old and fresh wounds are all susceptible to infection. There are a number of ways in which the tissue spaces of old wounds and ulcers, which are agglutinated or closed by granulation tissue, may be opened.

Erysipelas is *lymphogenous* when the infection is carried by the lymphatics from deeper inflammatory foci. Most frequently it develops from a streptococcic lymphangitis at some distance from the infection atrium, and often accompanies or follows a subcutaneous streptococcic phlegmon. Often a subcutaneous phlegmon develops secondarily to an erysipelas. In rare cases in which the pus containing streptococci extends from an osteal, articular, or glandular focus and penetrates the skin a true erysipelas instead of the indistinct redness develops.

Hæmatogenous erysipelas is rare. It occurs in metastatic streptococcic infections, as a rule, associated with metastatic phlegmons, suppurative arthritis, etc.

Ectogenous erysipelas develops most frequently upon the face, leaving out of consideration the relatively rare wound erysipelas which follows operations and injuries. Hæmatogenous erysipelas may develop upon any part of the body, while the areas affected in lymphogenous

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[1] In a very few cases staphylococci (Jordan), typhoid bacilli (Rheiner) and pneumococci (Perls-Neelsen) have been found in an inflammation of the skin which resembled erysipelas clinically.

erysipelas depend upon the position of the deep inflammation. In Rogers's statistics, comprising 597 cases, the face was involved 496, the face and scalp 96 times.

Infection Atria.—Chaps, fissures, rhagades, eczema, and small wounds, particularly about the nose and lips, afford abundant opportunity for infection. Furthermore, it is possible that infection may develop from the nasal secretion which contains streptococci, or may be produced by contact and rubbing with soiled fingers. Erysipelas of the mucous membranes of the nose, pharynx, mouth or lacrimal ducts, and often of the auditory meatus may extend to the face.

Character of the Inflammation, Clinical Course, and Symptoms.—An acute serous inflammation follows a streptococcic invasion of the cutis. The serous exudate sometimes becomes purulent. The subcutaneous tissue is involved in different degrees. There is a marked hyperæmia, a serous exudate is poured out, and the leucocytes migrate into the tissue spaces. The streptococci multiply and spread in the lymphatic vessels and spaces; the lymphatic vessels about the periphery of the inflamed area being filled with streptococci. They are found in the blood only in the hæmatogenous form or when a general infection develops. There is but little destruction of tissue in the nonsuppurative erysipelas. It runs its course in a few days or a week, and leaves no tissue changes other than desquamation and falling out of the hair. The latter results from exudation into the hair follicle. The hair rapidly grows again. Necrosis of the skin occurs only in the severer forms or in parts of the body (over shin, patella, olecranon, malar bone, spines of ilium) where there is but little subcutaneous fat and the skin is not distensible, or where the connective tissues are loose and permit of a large exudation, as in the eyelids. Necrosis of the skin occurs more frequently in the suppurative, phlegmonous forms.

At the beginning of an erysipelas the areas of skin involved are reddened, hot, indurated, and painful upon pressure and contact. The patient complains of a burning or stinging pain. A chill and high fever precede or accompany these local changes. In a few hours a sharply delimited redness and swelling of the skin develops. The rapidly extending borders of the swelling follow, according to Pfleger, the parallel linear furrows and connective tissue bundles of the corium, mostly in the direction of the lymph stream, but also against it. It sends out deep red, curved offshoots, flamelike tongues and points, between which lie pale areas of skin which were first affected. The inflammation rarely extends symmetrically, but more frequently from this and now from that border, just as the troops are thrown into a breach when storming a fortress. The inflammation may extend slowly or so rapidly that in a single night the entire face may be involved. Often the erysipelas

comes to a standstill in regions where the skin is firmly attached (e. g., to the crest of the ilium). Often large areas of skin are uninvolved and completely surrounded by the inflammation, so that they appear as white islands surrounded by reddened, erysipelatous skin. A marked œdema develops in erysipelas of the eyelids and external genitalia, as the underlying connective tissue is very loose.

Clinical Forms of Erysipelas.—The clinical forms which have been described frequently pass over into each other. Erysipelas erythematosum is characterized by the marked redness of the skin, erysipelas bullosum by vesicles, erysipelas pustulosum by pustules, erysipelas phlegmonosum by subcutaneous suppuration, erysipelas necroticum (gangrænosum) by necrosis of the skin. It is simpler, however, to speak of a suppurative and nonsuppurative form, for suppuration means, as a rule, not only a local, but also usually a general aggravation. With the extensive destruction of skin and subcutaneous tissues, as in severe phlegmons, there come the dangers of metastatic infection, favored by thrombophlebitis, and of general infections.

Erysipelas, as a rule, runs its course in from six to ten days. Its duration varies between hours and weeks. An erysipelas of the extremities, occurring with a lymphangitis, which is the most marked feature of the clinical picture, may disappear in a day. Erysipelas of the face and scalp does not, as a rule, last longer than a week. Only the rarer forms which migrate from the face, scalp, or extremities to the breast and back, and erysipelas of the trunk, last longer. There are cases of erysipelas in which the inflammation extends almost over the entire body, recurs in areas which have been healed for some time, persists for weeks, and exhausts the patient.

Recurrent and Habitual Erysipelas.—A recurrence of erysipelas, by which is understood a new infection and not a relapse occurring in the course of an existing inflammation, occurs where an open wound affords an infection atrium. Such infection atria are afforded by tuberculous fistulæ, ulcers of all sorts (syphilitic, carcinomatous, and varicose), rhagades and chronic eczema. An attack of erysipelas never affords an immunity. Streptococci, although of lessened virulence, which persist in such ulcers, invade the tissues again as soon as a mechanical irritation opens the lymphatic spaces of the cutis. Many individuals are subject to what is described as habitual erysipelas, which involves most frequently the face. In these cases the slightest irritation, such as sneezing or wiping the nose, may provoke an attack. These individuals have apparently but little resistance against streptococci.

Involvement of Lymph Nodes, Abscess Formation, Phlebitis, etc.—Redness and swelling of the skin are not the only symptoms of erysipelas. The neighboring lymphatic glands become painful and swollen early,

and in erysipelas of the extremities there is frequently an accompanying lymphangitis. Both usually subside without suppuration. Abscess formation is the exception.

Abscesses occur only in the suppurative form of erysipelas, and, as a rule, are produced by secondary infection with staphylococci. Phlebitis occurs especially in erysipelas of the legs. Thrombi then form which may suppurate if the vein wall is penetrated by cocci, and metastatic inflammation may develop as a consequence. Besides, there are general symptoms, different complications, and sequelæ.

Onset and Clinical Course.—The disease and the general reaction begin, as a rule, with a severe chill and fever. Frequently the temperature reaches 103° or 104° F. If the erysipelas extends, a continuous fever persists during its course, which falls by crisis or lysis as the inflammation approaches its termination. The fever is intermittent or remittent when the inflammation extends at irregular intervals (*vide* Fever, p. 164). The other symptoms of fever—rapid pulse, gastric and psychic disturbances, headache, restlessness, stupor, delirium—are frequently present in different degrees. For the most part these symptoms as well as the temperature are the result of the absorption of bacterial toxins. In erysipelas of the head and face the cerebral symptoms are frequently pronounced. They may pass into the symptoms of meningitis or may lead to the diagnosis of this complication. Fortunately meningitis follows but rarely erysipelas of the scalp and the secondary orbital phlegmon, by extension of a thrombophlebitis. In rare cases erysipelas runs its course without fever.

Bacteria but Rarely Found in the Blood in this Disease.—Streptococci are but rarely found in the blood (Pfuhl, von Noorden, Heitz, and Widal). Apparently they are retained in the lymphatic glands for the most part. The streptococci which do pass into the blood are rapidly destroyed. Even after death bacteria are not frequently found in the blood. Only in the severer forms of erysipelas and in those forms associated with metastatic inflammation are they found in the blood in large numbers.

Sometimes other bacteria besides the streptococci are found in the metastatic foci. These gain entrance through ruptured vesicles and necrotic cutaneous areas. Zeller and Arnold have found, for instance, a long, gas-producing bacillus.

Complications.—The complications of erysipelas result from an extension of the inflammation and from the development of metastatic infections. The inflammation may extend and produce a suppurative inflammation of the parotid gland, tendon sheaths, bursæ, muscles, and joints. A phlegmon of the orbital fat may develop and cause secondary suppuration of the bulb or meningitis. An acute inflammatory laryngeal

stenosis may follow the extension of a pharyngeal erysipelas to the larynx. This may extend and involve the lungs, which may also be infected through the blood stream. Pleurisy, which is rare, develops from foci in the lungs, or as a metastatic infection; endocarditis is rare.

Cardiac weakness, which may persist for a long time, is the most important of the sequelæ. It must be attributed to the action of the streptococcic toxins, although myocarditis is rare. The acute nephritis lasts but a short time, and only in case of preëxisting nephritis does it pursue a severe, occasionally a fatal course. Frequently a recurring erysipelas produces a chronic irritation of the skin which excites connective tissue growth. The lymphatics become occluded, and a lymphstasis or lymphatic œdema results (pachydermia of the face, Friedrich, Bernhardt, elephantiasis of the extremities and external genitalia).

Prognosis.—The prognosis is most favorable in the usual nonsuppurative erysipelas of the face and head, less so in erysipelas of the trunk, because of its longer course. In the suppurative forms general and metastatic infections develop most frequently.

Erysipelas in alcoholics, in patients weakened by previous disease, and in the newborn gives the most unfavorable prognosis.

Next to the cardiac weakness, pneumonia and meningitis are the most frequent causes of death, which, according to Zuelzer's statistics, comprising 10,000 cases, occurs in eleven per cent of the cases.

Diagnosis.—The diagnosis of erysipelas is, as a rule, easily made. The progressive and sharply limited redness and swelling of the skin and the high fever, which begins with a chill, are so characteristic that there can be but little doubt as to the diagnosis. The scalp shows but little redness, but the extension of the disease and the pain elicited by pressure indicate the nature of the inflammatory process.

Treatment.—The methods of the treatment, which are intended to control the disease, are as numerous as they are useless. There is no agent which will arrest the inflammation. Serum therapy has as yet been unsuccessful (*vide* Streptococci). Attempts to limit the extension of the inflammation by closing the lymphatic channels (cauterization of surrounding healthy skin, application of adhesive strips, collodion, and other agents) are unsuccessful, for the streptococci then seek the deeper lymphatics. Antiseptics, which have been injected into the inflamed area and into the healthy surrounding area, do not prevent the extension of the inflammation. Just as little is to be expected from the use of antiseptic compresses, applied with or without previous incision or scarification of the inflamed area and of antiseptic ointments. The ice bag and cold compresses should not be used, because of the danger of necrosis of the skin; multiple punctures are indicated to prevent the latter where the œdema is marked (eyelids).

The main object of the treatment should be to control the pain and prevent harm (secondary infection, mechanical irritation). Moist compresses and ointments lessen the pain. The former, however, almost invariably produce an eczema which provides new infection atria; non-irritating ointment dressings of vaseline and zinc oxid are therefore most useful. Gauze masks may be made for the face. The extremities should be immobilized by splints and elevated after application of the ointment dressings. It is understood that the patient should remain in bed. The suppurative forms of erysipelas should be treated according to the general principles already described (*vide* p. 197).

In habitual erysipelas a special treatment is demanded for the ulcers which afford the infection atria. Eczema should be treated with ointments, tuberculous ulcers by cauterization, other ulcers with suitable dressings, etc.

General treatment should combat the cardiac weakness. Nutritious food, wine, and injections of camphor should be given.

The method of treatment of wounds employed at the present time is the best prophylaxis. Streptococci are rarely found in the erysipelatous blebs (Respinger), so there is no more danger of contagion than in other suppurative infections. The disease follows the transference of the secretion containing streptococci to the infection atrium. For this reason patients with erysipelas should be placed in the isolation ward with patients suffering from acute suppurative inflammation (*vide* General Rules for Treatment of Suppurative Inflammation).

The therapeutic action of erysipelas upon malignant tumors and syphilitic and tuberculous granulation growths has attracted considerable attention lately. Busch (1866) demonstrated this action upon a sarcoma of the skin. The tumor became hyperæmic, underwent a rapid fatty degeneration, and disappeared by absorption following an attack of erysipelas. Clinical experience and the infection of patients suffering from inoperable tumors have demonstrated that the results are not sure and constant (Fehleisen, P. von Bruns; cf. also Tuberculosis, Part III).

[The mixed toxins of erysipelas and prodigiosus of Coley have been employed with curative effect in a small proportion of inoperable sarcomas.]

LITERATURE.—*M. Bernhardt.* Pachydermie bei habituellem Gesichtserysipel. Münch. med. Wochenschr., 1897, p. 887.—*P. Bruns.* Die Heilwirkung des Erysipels auf Geschwülste. Beitr. z. klin. Chir., Bd. 3, 1888, p. 443.—*W. Busch.* Einfluss von Erysipel auf organische Neubildungen. Berl. klin. Wochenschr., 1866, p. 245.—*Fehleisen.* Untersuchungen über Erysipel. Verhandlungen der Würzb. phys. med. Gesellsch. Sitzungsberichte, August, 1881, p. 126;—Das Erysipel. Deutsche Zeitschr. f. Chir., Bd. 16, 1882, p. 391;—Die Aetiologie des Erysipels. Berlin, 1883.—*Franke.* Ein Beitrag zur Frage der Kontagiosität des Erysipels. Deutsche Zeitschr. f. Chir., Bd. 78, 1905, p. 182.—*Friedrich.* Pachydermie im Anschluss an habituelles Gesichts-

erysipel. Münch. med. Wochenschr., 1897, p. 33.—*Jordan.* Ueber die Aetiologie des Erysipels u. s. w. Münch. med. Wochenschr., 1901, p. 1371.—*Klemm.* Ueber das Verhältnis des Erysipels zu den Streptomykosen. Mitteil. aus den Grenzgeb., Bd. 8.—*Köster.* Behandlung des Erysipels mit Vaseline. Therapeut. Monatshefte, 1896.—*Lenhartz.* Erysipelas und Erysipeloid. In Nothnagels spez. Path. u. Ther. Wien, 1899.—*v. Noorden.* Ueber das Vorkommen von Streptokokken im Blute bei Erysipelas. Münch. med. Wochenschr., 1887.—*Pfuhl.* Ein Fall von Allgemeininfektion mit Streptokokken infolge Hauterysipel. Zeitschr. f. Hygiene und Infektionskrankheiten, Bd. 12, 1892, p. 517.—*Respinger.* Untersuchungen über die angebliche Kontagiosität des Erysipels. Beitr. z. klin. Chir., Bd. 30, 1901, p. 261.—*Tillmanns.* Erysipelas. Deutsche Chir.

ERYSIPELOID

There is a disease, the local symptoms of which resemble closely those of erysipelas. It was known earlier as chronic erysipelas, erythema migrans, and was called erysipeloid [1] by Rosenbach. It develops most frequently from small wounds of the fingers, but is occasionally seen upon the nose, cheeks, and neck.

Onset and Clinical Course.—It begins with a mild burning and itching of the skin without fever or any general reaction. The skin becomes somewhat swollen, painful, and discolored a deep bluish red. It extends slowly from the infection atrium, the older area becoming pale, toward the hand, from the base of a finger to the neighboring finger, but rarely as high as the middle of the hand. A lymphangitis of the arm which resists treatment is seen in ten per cent of the cases.

The disease lasts usually one week. Many cases, however, namely, those which have not been treated, persist for three or four weeks.

Erysipeloid has some relation to dead, decomposing animal matter. It attacks frequently cooks, butchers, tanners, fishmongers, men who open oysters, and merchants who come in contact with cheese or herring. Almost always some wound can be demonstrated which affords the infection atrium.

Organism Found in Erysipeloid.—A cladothrix-like micro-organism was obtained in pure cultures from a diseased area of skin by Rosenbach (1887); inoculation of a cutaneous wound with this micro-organism caused erysipeloid. The findings have been confirmed by the researches of Ohlemann (1904). It is difficult to classify this irregularly round microbe, which develops into threads in old cultures.

Diagnosis.—It is impossible to mistake the disease when fully developed. Erysipelas extends more rapidly and is almost always accom-

[1] The author cannot accept the case reported by Tavel, which presented fever, general reaction and severe local symptoms, as one of pure erysipeloid. He sees yearly 30 to 40 cases of erysipeloid among the 10 to 20,000 patients treated at the Royal Polyclinic at Berlin. Apparently this case was one complicated by a pyogenic infection.

panied by fever. The redness associated with lymphangitis reticularis occurring upon the fingers has indistinct, never sharply defined boundaries.

Treatment.—The best and simplest treatment consists of immobilization (papier maché splint) of the fingers, to which vaseline has been applied, for two or three days. The redness rapidly fades, but if movements are made too early it recurs again in some areas. Resistant cases are rare.

LITERATURE.—*Cordua.* Zur Aetiologie des Erythema multiforme. Deutsche med. Wochenschr., 1885.—*Delbanco.* Ueber das Erysipeloid. Deutsche Medizinalzeitung, 1898, No. 78.—*Gilchrist.* Erysipeloid (329 Fälle). Journ. of Cutaneous Diseases, 1904, November.—*Ohlemann.* Beitr. z. Kenntnis des Erysipeloids und dessen Aetiologie. I.-D. Göttingen, 1904.—*Rosenbach.* Ueber das Erysipeloid. Chir.-Kongr. Verhandl., 1887, II, p. 75.—*Tavel.* Das Erysipeloid. Deutsche Zeitschr. f. Chir., Bd. 61, 1901, p. 528.

(b) THE PYOGENIC INFECTIONS OF MUCOUS MEMBRANES

The rich bacterial flora of the mucous membrane, which comprises not only harmless bacteria but pyogenic and putrefactive bacteria as well, may be easily increased during respiration or the taking of food. For this reason in many inflammations of mucous membranes mixed and secondary infections occur.

The pyogenic infections can, in spite of the many transitions, be differentiated from the putrefactive forms, in which putrefactive bacteria are the deciding factors.

Infection Atria.—Small injuries and large wounds, changes produced by diseases (diphtheria, gonorrhea, syphilis, tuberculosis, typhoid ulcers, cauterization, thrush, and ulcers due to dentition), or the anatomical relations of the mucous membranes covering lymphatic structures afford the infection atria. A lymphogenous inflammation develops when the infection travels through the lymphatics from a neighboring focus; a hæmatogenous inflammation may occur in general pyogenic infections, as a result of which small embolic abscesses may develop in the intestinal and gastric mucous membrane.

In certain diseases of the mucous membrane of the mouth cavity, which occur in chronic poisoning with mercury, phosphorus, lead, and arsenic, and begin with inflammatory swelling and exfoliation of the mucous membrane, the bacteria of the mouth cavity are able to invade the tissues, as their resistance is greatly reduced. In this way the bacteria participate secondarily in the severe ulcerating forms of inflammation associated with necrosis or gangrene.

Varieties.—The pyogenic bacteria found most frequently in inflammation of the mucous membrane are the staphylococcus, streptococcus,

gonococcus, pneumococcus, bacterium coli commune; to these may be added the bacilli of pneumonia and influenza.

The superficial inflammations of mucous membranes produce a serous or suppurative catarrh (from καταρρέω, to flow) or a fibrinous membrane (croupous inflammation). The mucous membrane becomes markedly hyperæmic and œdematous; in the larynx this œdema may be great enough to produce a dangerous stenosis.

A serous, purulent, or purulo-hæmorrhagic exudate is then discharged upon the surface of the mucous membrane, the normal mucous secretion of which is altered. Often in the mouth and pharynx vesicles are formed with resulting exfoliation of the superficial epithelial layers (desquamative catarrh), and superficial ulcers (catarrhal ulcers) which heal by granulation tissue form. The lymphoid organs are always enlarged and may suppurate (tonsillar abscess, follicular abscess, intestinal ulcer).

A fibrinous membrane is formed by the coagulation of the exudate, where the connective tissues are exposed after the destruction of the epithelium by inflammation or injury. The whitish yellow (if mixed with blood, brown) more or less firmly attached membrane shows a marked contrast to the reddened surrounding tissue. It resembles the pseudomembrane of diphtheria, but the fibrinous network and necrosis of tissue never extends so deeply in croupous inflammation. This fibrinous (croupous) inflammation which occurs in the upper air passages in a number of diseases (measles, scarlet fever, whooping cough, pneumonia, typhoid fever, etc.), and in which pyogenic bacteria participate (streptococci), is called diphtheroid to differentiate it from the inflammation produced by the bacillus of diphtheria. Similar fibrinous inflammations occur in the bladder, vagina, and intestine. Frequently they become secondarily infected with putrefactive bacteria and then gangrenous ulcers develop. Small, round, painful, yellowish areas, surrounded by a red zone, are produced by this fibrinous inflammation associated with necrosis of the epithelium. These occur frequently in the mouth and are called aphthæ.

Deep inflammations of mucous membranes develop from wounds, about penetrating foreign bodies, or extend from the inflamed surface of the membrane.

They produce a marked inflammatory œdema and a phlegmon of the submucous tissues. The surface of the mucous membrane is involved in different degrees; it may present only a catarrhal inflammation, or may become necrotic.

Pus collects in bony cavities lined by mucous membrane, in hollow organs such as the gall bladder and appendix when the outlet is occluded by inflammatory swelling of the mucous membrane or by other causes (e. g., suppurative otitis media, empyema of the gall bladder, and processus vermiformis). If secondary infection with putrefactive bacteria,

which wander in from the mouth or intestine, occurs the pus becomes foul smelling and the mucous membrane becomes gangrenous.

Erysipelas of the mucous membrane is an acute inflammation of the surface membrane, combined, however, with a submucous and a deeper phlegmon. An accurate diagnosis can only be made when the inflammation extends to the skin, although it may be suspected because of its violent course with high fever and severe general symptoms. It occurs in the pharynx, nose, larynx, and upon the female genitalia, and recurs frequently where a chronic inflammation of the mucous membrane favors the growth and invasion of the streptococci.

Results of Inflammation.—The results of inflammation of mucous membranes differ. Catarrhal inflammation and superficial ulcers heal by absorption of the inflammatory exudate and proliferation of the epithelium, leaving no trace of the inflammation. A scar is found when a deep ulcer heals. Incomplete repair or the frequent recurrence of mild inflammation produces a chronic inflammatory condition which results either in thickening and induration with glandular hypertrophy and growth of the lymphoid tissue or atrophy of the mucous membrane.

Fever and general symptoms usually accompany the acute inflammations of mucous membranes. They may even be present in a very mild angina or enteritis. Their duration depends entirely upon the course of the local inflammation.

The extension of these pyogenic infections from the upper air passages to the lung, and the different infections of the mucous membrane of the gastrointestinal tract are of much less importance to the surgeon than the suppurative phlegmonous forms of inflammation, the secondary diseases of the lymphatic glands, and the general infections with bacteria.

The phlegmon of the mucous membrane carries with it many dangers. A suppurative inflammation of the floor of the mouth develops from a phlegmonous glossitis or suppurative periodontitis. It is accompanied by a marked infiltration of the tissues, extends between the muscle planes of the neck, and may cause an œdema of the glottis or a mediastinitis, which proves fatal.

A phlegmon may spread under the mucous membrane of the mandible or cheek and produce a meningitis unless controlled. The tissue of the tonsil is frequently the seat of small abscesses, which develop from the crypts, and the origin of phlegmons which extend to the peritonsillar tissues and soft palate. Phlegmons developing in the pharynx or œsophagus may gravitate in the loose tissues surrounding these structures to the mediastinum. Abscesses of the intestinal wall and submucous phlegmons of the pylorus may rupture into the free peritoneal cavity and produce a suppurative peritonitis; this occurs most frequently in the ap-

pendix. Abscesses of the bladder may rupture externally and produce perivesicular suppuration, phlegmons of the urethral mucous membrane (by ulceration, injury during catheterization) may spread to the scrotum and perineum. Periprocteal abscesses develop in the tissue surrounding the rectum. These rupture externally and leave frequently the resistant fistulæ in ano. Where the submucous tissues are closely connected with bone, suppurative periostitis, osteomyelitis, and necrosis develop secondary to the phlegmon.

An inflammatory enlargement of the neighboring lymphatic nodes follows inflammation of a mucous membrane. The acute lymphadenitis occurring in the neck (submaxillary region in angina) is a well-known and striking example.

The glandular enlargement disappears as the inflammation subsides. This enlargement persists if the inflammation recurs frequently or if there is a chronic inflammation of the mucous membrane. In such cases chronic irritation leads to a hyperplasia of the glandular tissue. The glands suppurate only in the more severe forms of suppurative catarrh, in phlegmons and erysipelas of the mucous membrane.

The absorption of very virulent bacteria from diseased mucous membranes gives rise to metastatic inflammation or general infection. This may occur in superficial as well as in deep inflammations. It is well known that malignant and fatal general infections are produced by the streptococci, which enter the circulation from catarrhal, phlegmonous, and putrefactive inflammations of mucous membranes. In rare cases the colon bacillus may enter the blood during an enteritis. Metastatic inflammations occur much more frequently than the general infections. Streptococcic, staphylococcic, pneumococcic, and other infections may develop after an acute angina; inflammation of the accessory sinuses of the nose stands in intimate relation to suppurative arthritis, muscle abscess, metastatic phlegmon, and osteomyelitis.

Treatment.—No agent or measure should be employed in the treatment of pyogenic inflammations of mucous membranes which favor the absorption of bacteria. Mechanical irritation, such as painting the pharynx, irrigating the nose, antrum of Highmore, the urethra, etc., wiping, tearing, or curetting away the fibrinous (croupous) membrane, formerly extensively employed, does this.

Antiseptics do not retard the development of the bacteria imbedded in the mucous secretion, neither do they destroy those hidden in the folds and pockets of the mucous membrane.

On the other hand, if too strong they irritate the mucous membrane, destroy the surface epithelium, and in this way provide new infection atria. Besides, in washing the mouth small amounts of these antiseptics (e. g., potassium chlorate) may be swallowed and do harm.

The most important thing in the treatment of acute inflammations of mucous membranes is to remove mechanically the bacteria contained in the secretion or resting up the surface of the mucous membrane. This is done by frequently washing (depending upon location, by gargles, mouth washes, weak irrigation) with lukewarm water, physiological salt solution or very dilute antiseptic solutions (e. g., potassium permanganate, 0.5–1.0 : 2,000; boric acid, 0.5 : 1,000; sublimate, 0.5–1.0 : 5,000; besides acetate of aluminum, menthol, thymol, salicylic acid, etc.). A two to ten per cent solution of hydrogen peroxide with the addition of salt solution (for mouth wash and gargle) has become very popular. It has a deodorizing action and as the foam develops (free oxygen) it cleans the surface mechanically.

The hygiene of the mouth cavity is very important in preventing the extension of the inflammation from its mucous membrane and adjacent areas. Less importance should be attached to tooth pastes, soaps, and tinctures, than to the much more important mechanical cleansing with toothbrushes and mouth washes.

Phlegmons of the mucous membrane are treated according to general rules. Complications are treated according to their indications.

The use of iodoform gauze is recommended for resistant ulcers, where these are accessible. Where this is impossible, they may be painted with iodoform glycerin emulsion (von Mikulicz). Alcohol, camphor spirits, strong caustics, and the actual cautery may be used if necrosis and gangrene develop. In all severe infections the general nutrition and conditions should be improved, for in this way the local resistance is increased.

In chronic inflammation one per cent salt solution, mineral water, such as Emser and Seltzer water, the latter warm or mixed with milk and used as a drink, may be employed for gargles and inhalation. One per cent tannin and alum solution, two to ten per cent silver nitrate and iodin glycerin solution may be applied with a brush or cotton swab. In hypertrophies of the mucous membrane a concentrated solution of silver nitrate, tannin, etc., may be used. Enlarged palatal and pharyngeal tonsils should be removed by operation, as they may give rise to recurrent inflammation.

LITERATURE.—*Askanazy.* Enteritis phlegmonosa. Zentralbl. f. allgem. Path., 1895, p. 313.—*Feder.* Die Desinfektion der Mundhöhle. I.-D. Jena, 1900.—*Hasslauer.* Die Bakterienflora der gesunden und kranken Nasenschleimhaut. Zentralbl. f. Bakt., Bd. 33, Origin., 1903, p. 47.—*Heymann.* Handb. der Laryngologie und Rhinologie. Wien, 1899.—*Kraus.* Die Erkrankungen der Mundhöhle und der Speiseröhre. In Nothnagels Handb. d. spez. Path. u. Ther., Bd. 16.—*v. Mikulicz* und *Kümmel.* Die Krankheiten des Mundes. Jena, 1898.—*Miller.* Die Mikroorganismen der Mundhöhle. Leipzig, 1892.—*Stöhr.* Ueber die Lymphknötchen des Darmes. Arch. f. mikr. Anat., Bd. 33, 1889, p. 255.

(c) PYOGENIC INFECTIONS OF LYMPHATIC VESSELS AND NODES

Bactericidal Action of Lymphatic Tissue.—Bacteria and their toxins are rapidly absorbed by the lymphatics from infected wounds and ulcers. The endothelium lining the lymphatic vessels may be injured by bacteria and their toxins, leading to the formation of a thrombus such as occurs in thrombophlebitis, which will be described later. While in the lymphatic vessels and nodes the bacteria which have been carried by the lymphatics are exposed to the bactericidal substances of the tissue fluids, and unless present in large numbers or very virulent they are destroyed. Under certain conditions the bacteria may pass through this lymphatic barrier and produce in this way a general infection. Absorption is accompanied by marked symptoms only when the bacteria are so numerous or so virulent that they are not destroyed by the tissue fluids, or when their endotoxins which are freed during bacteriolysis cause inflammation. If the bacteria are numerous and highly virulent the tissues in which the lymphatics arise and those composing the walls of the lymphatic vessels and the lymph nodes react to the invasion, and an inflammation develops which retards or prevents the deposition and multiplication of the bacteria. Frequently the bacteria extend beyond the lymphatics and invade the surrounding tissues.

Blood infections occur after lymphatic involvement only when the bactericidal properties of the lymph nodes have been so reduced that they no longer offer a barrier to the extension of the bacteria into the larger lymphatic vessels.

The more virulent the bacteria the earlier the lymphatic vessels and nodes become inflamed. Lymphatic involvement may be exceedingly rapid after injuries received during post-mortem examinations or operations, for the pus found in fresh cadavers and in very sick patients frequently contains the most virulent bacteria.

Varieties.—Different varieties of pyogenic bacteria, especially staphylococci and streptococci, are the most frequent cause of acute, more rarely of chronic lymphangitis. Lymphangitis develops from recent infected wounds, from suppurating wounds, ulcers, and granulating surfaces, from superficial and deep inflammatory foci and inflamed mucous membranes.

The *local symptoms* of ACUTE LYMPHANGITIS are most striking when the superficial lymphatics of the skin and subcutaneous tissues are involved. Lymphangitis occurs most frequently upon the extremities, especially upon the arms, as wounds of the hand, which provide the infection atrium, are very common. When infection occurs at the points of origin of the lymphatic vessels (e. g., about a wound or excoriation of the little

finger or a furuncle of the arm), there develops a marked redness of the skin, the borders of which are always indistinct and extend in the direction of the lymph stream. Sometimes the inflammation, which is accompanied by an itching and burning and a sensation of fullness, develops without any apparent cause, frequently after mechanical irritation of the small wound. The redness may be diffuse, mottled or netlike, corresponding to the form of the plexus of capillaries, but in a few hours a number of red streaks develop from the reddened area. These gradually fuse, forming one or more streaks which correspond to the main lymphatic trunk or trunks which empty into the painful swollen lymph nodes of the axillary fossa, lying along the axillary vessels. The streaks developing in a lymphangitis of the foot extend toward the popliteal fossa or upon the anterior and medial surface of the thigh, where they end in the inguinal lymphatic nodes.

In from one to two days the redness of the streaks becomes deeper and the lymphatic vessels impart the sensation of hard cords, which are painful when palpated. The skin of the extremity involved becomes moderately swollen, painful, and tense.

Inflammation of the deep lymphatic vessels is indicated by a dull, distressing, rapidly increasing pain, and by swelling of the lymph nodes into which they empty. Frequently the deep lymphatics are involved alone or earlier than the superficial.

The *clinical course* of lymphangitis is sometimes mild, at other times severe, depending upon the bacteria concerned, the character of the inflammation, and the complications.

A superficial lymphangitis may subside in from one to two days, nothing remaining but a slight hyperæmia and a sero-cellular infiltration of the adventitia and adjacent tissues, which rapidly disappear. The epithelium covering the area involved exfoliates. In this simple form of lymphangitis new streaks, indicating the involvement of other lymphatic vessels, may develop for several days, while the ones which have developed earlier become pale and disappear.

In other cases the old streaks become transformed on the second or third day into hard cords the size of the little finger. In these cases the walls of the lymphatic vessels become hyperæmic, and an exudate is poured out into the tissues composing and surrounding them. The endothelial cells lining the vessels become swollen and are cast off, and thrombi of different lengths containing lymphocytes and endothelial cells are formed by the coagulation of the lymph. Thrombus formation usually begins about the valves of the lymphatic vessels, and when the larger vessels are closed (thrombo-lymphangitis) a stasis of lymph develops.

Involution is slow, requiring from one to two weeks, and while it is occurring the streaks become brown, then yellow, the œdematous swelling

and the hard cords disappear, for as the hyperæmia subsides the thrombi soften and become absorbed, the vessels become patent, the endothelium forms again, and the exudate is absorbed.

The suppurative lymphangitis is the most severe form. The inflammation then spreads from the hard cords, invades surrounding tissues, and produces subcutaneous abscesses. The thrombi undergo septic softening, and the vessel walls become necrotic. Abscesses develop about the lymphatic vessels, from which subcutaneous phlegmons may originate in the first or second week. One abscess develops from another after long intervals, and in this way the inflammation may extend over long periods.

The same changes occur in the deep lymphatic vessels when inflamed, but only the suppurative form gives rise to distinct symptoms when the inflammatory exudate having become purulent reaches the skin.

Complications.—Phlegmons and suppurative lymphadenitis are the most frequent complications of lymphangitis. Inflammation of the subcutaneous and deep veins accompanied by thrombosis may develop by direct extension of the inflammation, especially when suppurative, from the lymphatics to the veins immediately adjacent. Metastatic inflammation, especially of the lungs, is much less frequent in lymphangitis than in thrombophlebitis and suppurative lymphadenitis; still it is possible for a part of a lymphatic thrombus to pass through a diseased gland, which no longer retains small emboli and bacteria, and to reach the heart.

Frequently a mild or severe lymphangitis is the beginning of a general infection, and not infrequently the development of red streaks in the skin, so characteristic of lymphangitis, is the first indication of a beginning erysipelas.

The severity of the *clinical course* usually depends upon the virulence of the bacteria and the resistance of the patient. Severe general infections not infrequently develop from wounds received during post-mortem examinations and operations. Severe forms of lymphangitis develop also in alcoholics, diabetics, and patients whose resistance has been reduced by some other infection.

Many cases are very resistant to treatment. The inflammation accompanied by thrombosis subsides slowly, and symptoms redevelop when some movement is made or injury received.

The *diagnosis* of acute lymphangitis is rarely difficult. The inflammation about the point of infection might be mistaken for a subsiding erysipelas or erysipeloid, the redness of which has no longer sharp boundaries. Lymphangitis of the superficial vessels might be mistaken for phlebitis, but the cords developing in the latter are much thicker; lymphangitis of the deep vessels for an inflammation developing from bone. The latter mistake is most apt to be made when the lesion develops upon the inner side of the arm, in the popliteal fossa and Scarpa's triangle.

The *treatment* demands absolute rest of the entire extremity, which should be obtained by a loosely applied splint, and elevation maintained as long as any red streaks are to be seen or any cords to be felt. Muscular movements, rubbing and massage, which drive the lymph onward, carrying with it bacteria and particles of thrombi, favor the development of general infection and are to be avoided.

An ointment usually controls the pain. Abscesses should be incised when they form. Constrictors should not be applied in incising an abscess if there is a thrombo-lymphangitis, as particles of the thrombus may be separated and forced into the circulation.

CHRONIC FORMS OF LYMPHANGITIS develop upon the extremities after repeated acute attacks, or when there is a continual absorption of infectious materials from an eczema, an ulcerated area of the skin, etc. After a time the vessels become closed as a result of the organization of a thrombus or cicatricial contraction of the vessels, and a lymph stasis develops. Chronic lymphangitis and frequently repeated attacks of erysipelas are important etiological factors in acquired elephantiasis or pachydermia. In the treatment of chronic lymphangitis an attempt should be made to remove the cause and to prevent the stasis of lymph. Elevation of the extremity, supporting dressings, and massage should be used.

The lymph glands (lymph filters) catch and retain dust, granules of coloring matter, the decomposition products of cells (e. g., pigment of red blood corpuscles), and of absorbed exudates and bacteria. Dust, pigment granules, etc., remain in the lymph nodes and incite merely tissue changes resulting in hyperplasia or cicatricial contraction. Bacteria, however, if viable and capable of multiplying, incite inflammatory changes, for both bacterial and animal toxins (e. g., snake venom) when absorbed by the lymphatics produce acute or chronic inflammations which may end in pus formation if pyogenic bacteria are present.

An inflammation of the lymph nodes occurs with every inflammation of the area which they drain. Sometimes only one lymph node becomes inflamed, at other times the entire chain. The lymphatic vessels carrying the bacteria are frequently not involved, at other times they are severely inflamed.

The bacteria are usually carried to the lymph nodes by the lymph, but hæmatogenous infections may also occur.

Lymphadenitis may pursue an acute or chronic course; the cervical, axillary, and inguinal lymph nodes being most frequently involved.

The simple form, LYMPHADENITIS SIMPLEX, is usually secondary to some mild inflammation of the area drained by the nodes or to a suppurative inflammation, which has subsided rapidly under proper treatment. In these cases many small lymph nodes may be palpated, besides the

one primarily involved, which never becomes larger than a walnut and is round, hard, and somewhat painful. As there is little or no periadenitis the nodes can be displaced on the underlying tissues and moved freely under the skin, which is not reddened. When the cause is removed the swelling subsides. If, however, the infection continues, the lymph nodes may undergo a chronic hyperplasia.

Upon section the surface of the involved node has a homogeneous, grayish red color. It is hyperæmic and infiltrated with a serous or serofibrinous exudate, which renders the capsule tense. Histologically there is an increase in the number of lymphocytes and leucocytes which have migrated through the walls of the blood vessels. The endothelial cells lining the lymph sinuses have been cast off. As resolution occurs the cells and the exudate are absorbed, the hyperæmia subsides, and the endothelial cells regenerate.

Suppurative lymphadenitis (lymphadenitis purulenta) is accompanied by a periadenitis. There are two forms clinically.

The first, which accompanies severe wound infections and is accompanied by pronounced general symptoms, may be called the sero-suppurative form. It is usually caused by streptococci, and has a tendency to form progressive, subcutaneous, and intermuscular phlegmons of the muscles and fascia of the neck, thorax, and abdominal wall. The swollen lymph nodes, which are painful to touch and upon motion, can no longer be distinctly palpated after a few days, as the inflammatory œdema of the surrounding tissues renders palpation of the separate nodes impossible. The skin, which is somewhat reddened, is adherent to a fairly hard, non-fluctuating mass, the size of a fist.

When exposed during an operation the individual nodes are seen in the œdematous surrounding tissues, agglutinated by a fibrinous periadenitis. Upon section small and large abscesses, gray necrotic areas and minute hæmorrhages may be recognized in the dark red parenchyma. Small abscesses may be found in the fat surrounding the lymph nodes. Microscopically the lymph sinuses are seen to be filled with and dilated by fibrinous masses, which contain leucocytes, red blood corpuscles, and innumerable bacteria. The latter are scattered throughout the node. If an acute progressive phlegmon has already developed, the nodes will be necrotic.

The second, which forms abscesses, may develop from the sero-suppurative form. As a rule, it pursues a mild course (subacute) from the beginning, and shows first the characteristics of a simple inflammation, and produces after the suppuration extends through its capsule (periadenitis purulenta) to the surrounding tissues, not the progressive, but the circumscribed phlegmon. Clinically in the beginning the nodes are hard and movable. Gradually they become fixed and adherent to

the reddened skin, soften in the center, fluctuate, and seem to be about ready to break through the skin. The single nodes which are agglutinated suppurate. The pus then breaks through the capsules of the separate glands and forms a large lymphadenitic abscess, which may discharge spontaneously. Fever and general symptoms are present, as a rule, in the beginning or during the development of periadenitic phlegmons. The inguinal lymphadenitis (or inguinal bubo) following the soft chancre belongs, as a rule, to this form of infection. In the pus of the bubo as well as in the secretion of the chancre is often found a chain-like bacillus (Ducrey, Krefting, Unna).

The bacteria most frequently found in lymphadenitis are the same as those in lymphangitis. Gonococci have been found in the so-called gonorrheal bubo. Mixed infections frequently occur. If infections with putrefactive bacteria occur the clinical picture resembles the sero-suppurative form, with the addition of the gangrenous destruction of the glands and their surrounding tissue.

It is not always possible to demonstrate bacteria in the inflamed nodes. It is easy where there is an acute inflammation which has extended rapidly to the surrounding tissues. If an abscess of a lymphatic gland has persisted for some time, the bacteria are frequently attenuated or dead. In a simple inflammation of a lymph node the bacteria are quickly killed.

The PHLEGMON has already been mentioned as one of the complications of an acute lymphadenitis. Where the glands lie directly over large veins (jugular, femoral, saphenous, axillary), a thrombophlebitis may develop. Pulmonary infarcts, followed by abscess, may develop secondary to the latter. The same may happen if the glands are mechanically irritated (rapid movements, trauma, massage), for their small capsular veins contain thrombi and their lymph sinuses coagulated masses of fibrin permeated with bacteria.

Emboli may be set free from these vessels, even after the symptoms of the inflammation have subsided. GENERAL INFECTION is another complication, for in the severe wound infections which are accompanied by lymphangitis, erysipelas, and phlegmons the lymph nodes are no longer able to arrest the virulent, incessantly developing bacteria.

The diagnosis of acutely inflamed lymph nodes is not difficult. The position and mode of extension of a fully developed phlegmon or abscess suggest that it has developed from lymph glands.

It is understood that in the TREATMENT attention must be paid to the primary focus. The diseased glands demand rest above everything else. Frequently the splint applied for the lymphangitis, phlegmon, etc., and simultaneously elevation of the extremity cause the simple inflammation of the gland to subside. Any movement, likewise any mechanical irri-

tation (by rubbing in of ointments, massage, pressure by means of sand bags, lead plates, which are intended " to distribute " the inflammation), is dangerous and should be given up, for it forces the bacteria and coagulated serum with the lymph or exudate into the surrounding tissues, if not into the blood.

Agents which produce a mild hyperæmia, such as mercury ointment, tincture of iodin, blur the local changes so that it is no longer possible to recognize the inflammation of the skin, and they are not superior to absolute rest obtained by an immobilizing dressing. The moist compress is of value in simple lymphadenitis, for example, in the neck, as it lessens the pain. In suppurative lymphadenitis the compress, as well as poultices, does harm (*vide* Fundamental Rules, p. 198).

The suppurative forms depending upon the indications are to be excised, incised, or aspirated. In the severe sero-suppurative forms all the inflamed nodes should be removed after being freely exposed. Simple incision is never enough, for a new phlegmon will develop from the remaining infected nodes. The lymphatic circulation is reëstablished even after the removal of all the nodes by a proliferation of the vessels in the surrounding fatty tissue (Bayer).

A wide incision is sufficient in the form which results in abscess formation, for the remaining glands are extruded and the inflammation does not extend. Incision is necessary when there is fever or a circumscribed phlegmon develops. If the abscess is limited to a group of nodes, puncture and removal of the pus is all that is required. Liquefaction of the nodes which have not softened frequently follows the injection of a small amount of a 1 per cent solution of benzoate of mercury, 0.1 per cent sublimate solution, or of physiological salt solution (Welander, Thorn, Wälsch). The injection treatment is especially valuable in the venereal bubo, as the patient is not compelled to remain in bed. It should be employed, however, only in softened glands, for the injected fluid raises the pressure in the gland and carries with it the same dangers as any other mechanical irritation.

Blood vessels have been injured in making incisions and punctures, because of carelessness or of ignorance of the anatomy of the part.

Rest in bed is necessary as long as there is fever.

The chronic inflammation of lymph nodes (lymphadenitis chronica simplex), like chronic lymphangitis, is produced by frequently recurring, mild inflammations of the periphery, or by the continual absorption of inflammatory substances from a chronically inflamed mucous membrane, an eczematous or ulcerated skin area.

The involved nodes are either enlarged and soft and present microscopically the picture of pure hypertrophy, or they are small and hard,

because of the hypertrophy and contraction of their connective tissue trabeculæ and capsule (fibrous hyperplasia).

Hypertrophied cervical glands accompany eczema, rhagades of the nose and lip, and catarrh of the mucous membranes. They may become tuberculous. If the nodes are adjacent to a carcinoma and are indurated they may be regarded as carcinomatous.

The treatment of chronic lymphadenitis usually depends upon the cause.

LITERATURE.—*F. Fischer.* Krankheiten der Lymphgefässe, Lymphdrüsen und Blutgefässe. Deutsche Chir., 1901.—*Thorn.* Behandlung der Leistenbubonen mit Injektion von Hydrargyrum benzoicum oxydatum. Deutsche med. Wochenschr., 1897, therap. Beilage, p. 49.

(d) THE PYOGENIC INFECTIONS OF BLOOD VESSELS

A suppurative inflammation of the walls of arteries and veins develops when an inflammation extends to the vessels from an adjacent focus or when the infection is carried by the blood. It occurs, therefore, in areas adjoining and in foci of inflammation, in general and embolic infections.

ARTERITIS PURULENTA begins as a peri- or as an endoarteritis, depending upon whether the bacteria enter the vessel from within or without. It is rarer than the corresponding inflammation of the veins, as the arterial walls are thicker and heavier. The pus of an acute or chronic abscess bathes for a long time the wall of a large artery which is separated from its surrounding tissue, and a periarteritis develops. This occurs often in the large, chronic abscesses of the neck and inguinal region, which have thrived upon treatment with poultices. Trauma is also a factor, for the suppuration is most marked where the arterial wall has been crushed during ligation (with infected ligatures), as in an injury, or where it has been pressed upon by a drainage tube, improperly placed.

Endarteritis develops if an arterial thrombus laden with cocci suppurates, or if an infected embolus lodges in the artery. Bacteria may pass with the blood stream through the vasa vasorum and lodge in the media and adventitia. Thrombosis with necrosis and gangrene then follow the inflammatory changes in the vessel wall (as in influenza, typhoid fever).

Mild inflammations produce merely a cellular infiltration and thickening of one or all of the tunics of the vessel wall. Inflammatory changes in the intima (endarteritis productiva) may result in obliteration of the lumen of the vessel; severe inflammation ending in suppuration destroys the vessel wall.

If necrosis of the suppurating infiltrated tissue occurs, the vessel wall becomes eroded and ulcerated. In the small arteries an obturating thrombus, which is destroyed if necrosis occurs, frequently prevents hæmorrhage. Severe hæmorrhage follows the rupture of the large branches and main trunks. This hæmorrhage (secondary) is to be feared, especially in large necrotic and gangrenous foci. It was the source of constant anxiety to the military surgeons of preantiseptic times in their amputations and disarticulations. If the vessel ruptures into an abscess cavity, a false aneurysm or a pulsating hæmatoma develops. The development of a true aneurysm frequently precedes rupture of the diseased vessel wall.

If the remaining tunic at the point of ulceration (the intima, if the suppuration extends from without, the adventitia if from within) is forced outward or its entire circumference is widened before being ruptured by the blood pressure, a spontaneous aneurysm is formed. If an infected embolus is the cause of this dilatation, the aneurysm is called embolo-mycotic. In order to prevent the dangerous arteritis of the large vessels in inflammatory foci, the surgeon should be careful in incising deep phlegmons and abscesses not to separate the connective tissue sheath of the large vessels. Where a ligated artery is exposed in a suppurating wound (e. g., a suppurating, therefore open amputation-wound) it should be supported for at least a week by a tampon, so that the full force of the pulse beat is not expended upon the arterial wall.

The development of an embolic aneurysm often indicates threatened rupture of the diseased arterial wall. For example, if a pulsating swelling, associated with severe local pain, develops within a few days in a patient suffering with endocarditis or general infection, and a few days before this sudden circulatory disturbances occurred in the extremity which made probable the diagnosis of embolism; double ligation of the vessel should be made, as in hæmorrhage, above the diseased area in healthy non-inflamed tissue. SUPPURATIVE PHLEBITIS (phlebitis purulenta) frequently begins as a periphlebitis associated with an inflammation of the lymphatic vessels surrounding or accompanying the large veins, or follows the extension of an acute suppurative inflammation from the cellular tissue surrounding the vein. If the inflammation develops from the lumen, thrombosis and infection through the blood stream precede it. In an inflamed area even the smallest veins are involved, for the inflammatory slowing of the blood stream and stasis favor the formation of thrombi and the growth of bacteria. Mild inflammations which do not end in suppuration produce, when subcutaneous veins are involved, painful hard cords of finger thickness which may be palpated under a reddened skin. The clinical picture resembles somewhat

that of thrombolymphangitis. These inflammations may subside completely; the thrombus is then organized or absorbed, the lumen becomes closed or patent. This may occur even in large vessels. Frequently recurring inflammation of veins (occurring in the leg with varicose ulcers) produces a chronic thickening of all the coats with a narrowing or obliteration of the lumen (phlebitis chronica hyperplastica). If the organizing thrombus becomes partially calcified, vein stones or phleboliths are formed. Severe inflammations lead to a purulent infiltration of the vein wall and surrounding tissues, and during the operation the yellowish, discolored, rigid, and thickened vein is found in suppurating or œdematous tissue. Venous thrombosis occurs constantly with suppurative phlebitis. The coagulation is produced by the bacterial toxins (Talke) which penetrate the intima, and by the inflammatory exudate which is poured out from the vasa vasorum (thrombophlebitis purulenta). The converse is true, that phlebitis follows suppuration of a thrombus. After suppuration of the thrombus and destruction of the vein wall, the pus escapes from the lumen of the vessel into the surrounding tissues and produces an abscess, a progressive suppurative inflammation, or becomes mixed with the exudate which is already present. Hæmorrhage rarely occurs, for the thrombus while softening has extended and has closed the vein proximally and distally. The diagnosis of thrombophlebitis of subcutaneous veins is not difficult. It may be mistaken for a thrombolymphangitis. The diagnosis of inflammation of deep veins may be made by the presence of œdema, by the palpation of hard, painful cords corresponding to the position of veins, severe pain, the presence of a local cause (such as varicose ulcer), and general symptoms. The following are the dangers which accompany thrombophlebitis:

An extension of the inflammation along the vein. A suppurative meningitis may follow a furuncle of the lip, as the inflammation extends along the facial to the ophthalmic vein and to the cavernous sinus. A phlegmon of the scalp extends to the veins of the diploë and dura, while inflammation of the umbilical vein may be the cause of a fatal peritonitis in the newborn. A thrombophlebitis of the veins of the puerperal uterus extends along the spermatic and hypogastric veins to the common iliac and femoral veins and the inferior vena cava. The inflammation extends with the growth of the thrombus against the blood stream. The veins of the mesentery become inflamed in perityphlitis and severe enteritis, and the inflammation may extend to the portal vein.

The second danger is the *separation of emboli which contain bacteria from suppurating thrombi.* Thrombophlebitis may thus become the cause of a metastatic infection. Any trauma or movement may separate or set loose an embolus in the small and large veins. As there are venous thrombi which contain bacteria in every inflamed area,

this danger must be kept in mind in the treatment of all pyogenic infections.

The treatment of acute thrombophlebitis demands in the first place absolute rest of the extremity involved. This is obtained by immobilizing dressings and rest in bed, which should be continued as long as there are any signs of inflammation. Abscesses should be incised. If chills, a high remittent fever, general symptoms, and those of lung embolism lead to the suspicion that a demonstrable thrombus is suppurating and breaking down, ligation and resection of the vein above the thrombus (if possible resection of the diseased portion, or at least removal of the suppurating thrombus) prevents in many cases general infection. The inflamed subcutaneous veins of the arm and leg (Lee, W. Muller) and the femoral vein (Kraussold) have been ligated with success. The internal jugular vein is ligated in thrombosis of the transverse sinus following suppurative otitis media (Zaufal), and the facial vein, when inflamed, secondary to carbuncle of the face. Trendelenburg records a case of general chronic puerperal infection which recovered after double ligation of the inflamed and thrombosed right hypogastric and spermatic veins.

LITERATURE.—*v. Büngner.* Spontanruptur der Art. femoralis. Arch. f. klin. Chir., Bd. 40, 1890, p. 312.—*Fr. Fischer.* Krankheiten der Lymphgefässe, Lymphdrüsen und Blutgefässe. Deutsche Chir., 1901.—*Frommer.* Zur Kasuistik der Nachblutungen. Arch. f. klin. Chir., Bd. 67, 1902, p. 439.—*W. Müller.* Zur operativen Behandlung infektiöser und benigner Venenthrombosen. Arch. f. klin. Chir., Bd. 66, 1902, p. 642.—*Nasse.* Mykot. Aneurysma der Art. femoralis. Deutsche med. Wochenschr., 1898, Vereinsbeilage, p. 259.—*Talke.* Experim. Beitrag zur Kenntnis der infektiösen Thrombose. Beitr. z. klin. Chir., Bd. 36, 1902, p. 339.—*Trendelenburg.* Ueber die chir. Behandlung der puerperalen Pyämie. Münch. med. Wochenschr., 1902, p. 513.

(e) PYOGENIC INFECTIONS OF BONE

Etiology.—Bone may be infected in three ways: 1. In compound fracture or in operations such as amputations, joint resections, and osteotomies, in which the medullary cavity or the surface of the bone stripped of its periosteum is directly exposed to infection. 2. A suppurative inflammation of the surrounding soft tissue may extend to the bone, and it may become involved secondarily. 3. The infection may be carried through the blood by bacterial or infected emboli, which lodge in parts of the bone where anatomical conditions are favorable or where a *locus minoris resistentiæ* has been provided by some previous injury or circulatory disturbance.

The classification of suppurative inflammation of bone is based entirely, or almost entirely, upon the tissues involved. Inflammation of the periosteum is called periostitis; of the bone marrow, osteomyelitis; of the

cortex, osteitis. Usually when all the different tissues of the bone are involved some collective term is required: osteitis as well as osteomyelitis being used in this sense.

Any pyogenic organism may be the cause of osteomyelitis. In the ectogenous infections, and those extending from inflammatory foci in the surrounding soft tissues, the staphylococcus aureus and albus and the streptococcus, often associated with other bacteria (e. g., putrefactive), are most frequently found. In hæmatogenous osteomyelitis the staphylococcus pyogenes aureus is found with by far the greatest frequency; then follow next in order of frequency mixed infections with the aureus and albus and the aureus and the streptococcus. The white staphylococcus and the streptococcus are more rarely found alone. The pneumococcus, typhoid bacillus, the gonococcus and bacillus of pneumonia are also found in osteomyelitic foci, but cases of osteomyelitis caused by these bacteria are relatively rare when compared to those caused by the bacteria above mentioned.

Pathology.—The pathological changes eventually are the same whether the inflammation attacks the bone from within or without, the order in which the tissues are involved being merely reversed.

If the inflammation develops from without, as occurs most frequently after an injury, or secondary to a phlegmon, a periostitis develops first, the periosteum becoming swollen and reddened and raised from the bone by a layer of pus. The inflammation next extends along the vessels of the Haversian canals, and the cortex of the bone becomes involved. In the short and flat bones an inflammation beginning in the periosteum frequently extends to the medulla. In a suppurative arthritis accompanied by a destruction of the articular cartilage, the spongy bone of the epiphysis is involved, and in com-

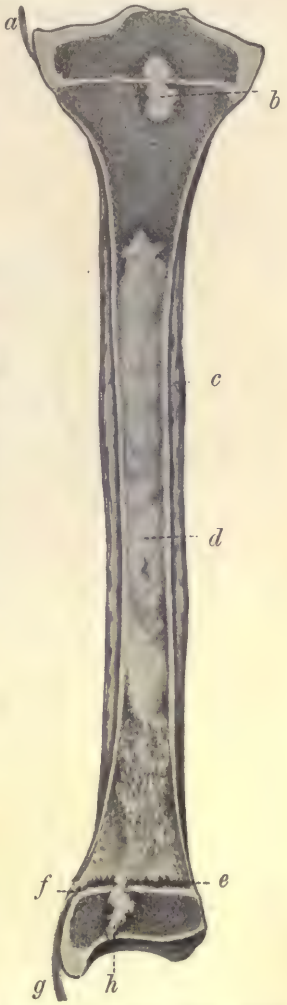

FIG. 101.—SUPPURATIVE OSTEOMYELITIS OF THE TIBIA (SEMIDIAGRAMMATIC). *a*, Attachment of capsular ligament; *b*, purulent focus in metaphysis which has ruptured into the epiphysis; *c*, periosteum raised by pus; *d*, phlegmon of the medulla; *e*, separation of the epiphysis; *f*, extracapsular rupture of pus; *g*, capsular ligament; *h*, rupture into joint.

pound fractures in which the medulla is exposed infection, if it occurs, travels rapidly along the medullary cavity.

In HÆMATOGENOUS INFECTIONS the medulla is usually primarily involved, occasionally the cortex and periosteum. When the infection begins in the medulla, it may travel in a number of different ways. The acute progressive infections of the medulla (medullary phlegmons) are frequently

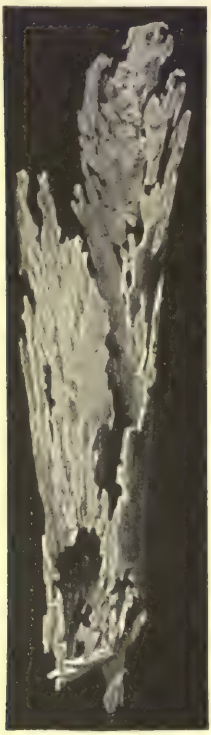

FIG. 102.—TUBULAR SEQUESTRUM.

FIG. 103.—TOTAL NECROSIS OF THE HUMERUS. Involucrum with sequestrum and cloacæ.

limited by the epiphyseal cartilages, but not infrequently the union between the metaphysis [1] and the epiphyseal cartilage is destroyed and the epiphysis becomes separated.

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[1] Metaphysis—a term used by Kocher to designate the spongy end of the diaphysis lying next to the epiphysis (Fig. 101). A suppurating focus situated in the metaphysis near the epiphyseal cartilage may produce: 1. A medullary phlegmon. 2. Extending along the epiphyseal cartilage, a separation of the epiphysis. 3. Passing

As the inflammation extends rapidly outward along the Haversian canals, the vessels of which become closed by thrombi as a result of the inflammation, a medullary phlegmon is usually accompanied by a suppurative periostitis of the same extent. The periosteum is raised from the bone by a thick layer of pus, which finally ruptures through it at a number of different points, where it becomes necrotic. The compact bone bathed in pus without and within, deprived of nutrition by the separation of the periosteum and thrombosis of the medullary vessels and those in the Haversian canals, becomes necrotic (necrosis totalis). If only the inner layers of the shaft or the deeper spongy bone becomes necrotic, one speaks of a central necrosis in contradistinction to the external or superficial necrosis, which occurs in periosteal or cortical suppuration.

FIG. 104.—TOTAL NECROSIS OF THE HUMERUS AS SEEN IN A ROENTGEN RAY PICTURE.

The bloodless white bone, killed by the suppurative inflammation and permeated with bacteria, excites and maintains in the living bone surrounding it a reactive inflammation which may be rarefying or demarcating as well as osteoplastic. Granulation tissue develops at the boundary between the living and dead bone from the healthy marrow, the spongy bone, and the Haversian canals. This tissue gradually develops to such an extent that the space (demarcation pit) between the dead (sequestrum) and the healthy bone is completely filled. The sharp and jagged form of an old sequestrum penetrated by

through the epiphyseal cartilage where pierced by canals for blood vessels an inflammation of the epiphysis. 4. Extending through the epiphysis a suppurative arthritis. 5. Passing along the epiphyseal cartilage and rupturing through the periosteum an intra- or extra-articular abscess, depending upon the insertion of the capsular ligament.

canals and traversed by grooves is due to the digestive action of the granulation tissue and not to the pus (von Volkmann). It differs from the even symmetrical absorption or necrosis of fresh macerating bone. The total and central sequestra of the diaphysis are cylindrical or tubular in shape, while cortical sequestra resemble a disk or chip. Weeks and months are required for a complete separation of a sequestrum, depending upon the extent of the necrosis. Often half a year is required for the separation of a large sequestrum of the diaphysis. If superficial, the separated sequestrum may be discharged with the pus when it ruptures externally, or it may remain and be digested and absorbed by the granulation tissue. Only very small sequestra, most frequently those derived from spongy bone, can be destroyed in this way.

During the separation and erosion of the sequestrum reparative processes leading to the formation of new bone are going on. The periosteum takes a very active, the medulla and surrounding intermuscular tissue a less active, part in this new bone formation. These reparative processes are most active in long hollow bones; least so in flat bones. Early, often within a week, the inner layer of the periosteum (cambium, germinal layer) begins to develop delicate layers of spongy bone (periostitis ossificans). This proliferation continuing gradually produces in the course of months a bony shell, which in the beginning is thin, fragile, and porous, like pumice stone. Later it becomes thicker, shapeless, and sclerotic, surrounding the dead bone or sequestrum like a capsule. This newly formed bone, which is separated from the sequestrum by a thin layer of granulation tissue deficient at some points, and pus, is called the involucrum (capsula sequestralis).

Canals of different sizes (cloacæ) lined with granulation tissue through which is discharged the pus forming in the interior are found in the involucrum. The involucrum is deficient and weakened where the periosteum has become necrotic, and if weight is brought to bear upon the bone or it is manipulated roughly the involucrum may be fractured. If the involucrum is fractured union may not occur, a pseudarthrosis developing. When the newly formed bone hardens the involucrum ceases to increase in size (von Volkmann). The spongy and compact bone may become so thickened and condensed as a result of the reactive inflammation, that the surrounding bone becomes as hard as ivory (osteomyelitis ossificans, scleroticans, eburnatio). This ossifying or sclerotizing process may involve an area 5 cm. in width surrounding a suppurating focus or a sequestrum in spongy bone.

This hard bone developing about a total sequestrum of the shaft may entirely fill up, or, as happens in the chronic sclerotizing forms of osteomyelitis, entirely obliterate the medullary cavity.

Clinical Course.—The clinical course and picture of suppurative osteomyelitis differ widely, the differences depending upon the virulence of the bacteria, the susceptibility of the tissues and of the patient, the location and predisposing cause, such as trauma, exposure to cold, etc., the complications (suppurative arthritis and metastatic infections), the predominance of necrotic or osteoplastic processes.

The hæmatogenous is the most important form of suppurative osteomyelitis. The way in which this form of osteomyelitis develops has been made clear by a number of very conclusive pieces of experimental work.

If a small amount of a virulent culture of staphylococcus pyogenes aureus is injected into the vein of a young rabbit or guinea pig, the

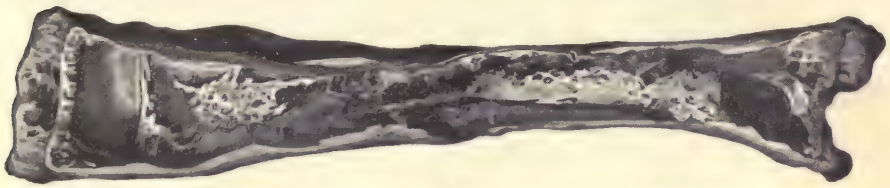

Fig. 105.—Tibia of a Young Rabbit with a Total Sequestrum of the Diaphysis, Developing Three Months After an Intravenous Injection of an Attenuated Culture of Streptococci. (¼ natural size.)

animal develops a fever and dies within a few days, and a post-mortem examination reveals numerous abscesses in the viscera, muscles, bones, and joints. The smaller the amount of the culture injected the more marked the development of abscesses in the bones, the less marked their development in other parts of the body. The abscesses developing in the bones are most commonly situated in the broad metaphysis of the femur, the upper end of the tibia, and in the upper extremity of the humerus. The disease procured in animals experimentally is, like severe osteomyelitis occurring in man, a fatal general infection accompanied by the formation of metastatic foci developing especially in bones (Rodet, Colzi, Lannelongue and Achard, Lexer).

Similar results may be obtained by the injection of the staphylococcus pyogenes albus, and the streptococcus pyogenes (Lannelongue and Achard, Lexer) and the bacterium coli commune (Ackermann).

If old attenuated cultures of the yellow or white staphylococcus are injected the animal remains sick for a short time, but recovers. During the course of the sickness several hot painful swellings develop upon one or more legs, and as the swelling of the soft tissues subsides the thickening of the bones, which after two or three weeks present all the pathological changes of chronic suppurative osteomyelitis (osteomyelitis purulenta chronica), as it occurs in man, becomes more distinct.

It is impossible to produce in animals by the intravenous injection of

staphylococci an acute progressive medullary phlegmon, unless an inflammation has been produced by the previous injection of some other organism, e. g., a pyogenic bacillus which occurs frequently in rabbits (Lexer).

Trauma has an actual influence in determining the location of and contributing to infections in experimental animals. Extensive suppuration develops at the seat of fractures or where the bones have been injured after the intravenous injection of virulent cultures made at the same time or some days later (Ullmann).

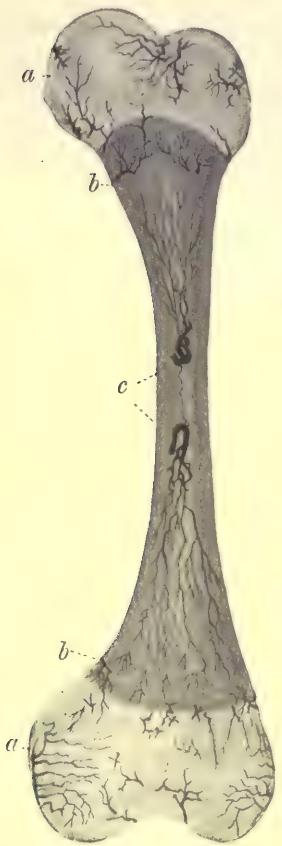

FIG. 103.—FEMUR OF A CHILD FOUR WEEKS OLD, THE VESSELS OF WHICH HAVE BEEN INJECTED, AS SEEN IN A ROENTGEN RAY PICTURE (PERIOSTEUM AND CAPSULAR LIGAMENTS DISSECTED AWAY). *a*, Epiphyseal arteries; *b*, metaphyseal arteries; *c*, double nutrient arteries.

Animal experiments have shown that pyogenic bacteria, and of these most frequently the staphylococci, may be deposited by the blood stream in young growing bones producing suppurating foci which are situated as in man in the metaphysis of the long, hollow bones, and in parts of the bones the resistance of which has been reduced (LOCUS MINORIS RESISTENTIÆ) by some trauma, exposure to cold, etc.

The micro-organisms may be absorbed from any inflammatory focus, no matter how small, or may be carried in emboli (infected emboli), or in groups (bacterial emboli), from the veins of the primary suppurating focus, which have become closed by thrombi. It may be impossible to demonstrate the primary focus, but a suppurating focus in bone is proof positive that a primary focus exists or has existed in each case (Jordan).

The absorption of bacteria, which may occur in any wound infection and bacterial invasion, leads to the development of a suppurative osteomyelitis only when special conditions are provided. The bacteria, unless present in large numbers or continually invading the blood, are deposited in the bone marrow, spleen and liver (Wyssokowitch), where they are exposed to the action of the bactericidal substances, which in the bone marrow are formed especially by the leucocytes. Here they are either killed or so injured by bactericidal substances that they can no longer multiply and invade the tissues (A.

Wassermann). In many infections the bone marrow is more active than any other tissue in producing the specific immune bodies, and therefore the deposition of bacteria in it may be regarded in the light of a protective measure, as the bone marrow destroys the bacteria and produces substances which are at the disposal of the organism in combating infections.

E. Fraenkel, and earlier Weichselbaum, demonstrated in the bone marrow of patients dying of pneumonia, felons, phlegmons, and erysipelas, the bacteria which had produced these lesions even when there had apparently been no blood infection. One must conclude, therefore, that bacteria are often deposited in the bone marrow even when the local infection and the general reaction is not severe.

If the pyogenic bacteria are present in the bone marrow, one of two conditions must be fulfilled before they can produce suppuration. The bacteria must either be virulent enough or present in large enough numbers to resist the bactericidal substances, or the tissues must be so weakened by trauma or circulatory disturbances that they can no longer produce these substances in large enough amounts to restrain the growth of the bacteria (A. Wassermann).

Osteomyelitis may also be produced by the displacement and lodgment of infected or bacterial emboli from the primary focus, which always contains veins which have been closed by thrombi or invaded by bacteria. Osteomyelitic foci which are intimately related to the arterial branches and are situated in the epiphyseal zone to which capillaries from all sides converge, and the foci in the short and flat bones (e. g., vertebræ, pelvis, phalanges) most frequently attacked by tuberculosis, must be regarded as of embolic origin (Lexer). The vertebræ, pelvis, and phalanges, etc., are much less frequently the seat of suppurative than tuberculous lesions, and embolism (by infected or bacterial emboli) is apparently much less frequent in suppurative than in tuberculous osteomyelitis.

Suppurative foci are more frequent in the metaphysis of long bones than in any other bones of the skeleton. The frequency of the lesions in the metaphysis cannot be satisfactorily explained upon the supposition that the bacteria are attracted by the bactericidal substances, for theoretically the diaphysis is as rich in these as the metaphysis; neither can it be explained satisfactorily by the lodgment of emboli, for other bones should then be attacked as frequently. The mechanical conditions provided in the epiphyseal zone of growing bones, in which there is a physiological hyperæmia with a slowing of the blood stream, and by the arrangement of the smaller vessels and the capillary loops with their branches which pass down into the primary medullary spaces of the epiphyseal cartilage (Langer), favor the deposition and retention of bac-

teria and explain the frequency of acute suppurative lesions in this part of the bone.

The fact that staphylococci are retained in these vessels and cause suppurative osteomyelitis more frequently than any other variety of bacteria, must be ascribed to their peculiarity of growth, occurring as they do in groups or clumps. If such a group gradually forms in these vessels, or if a group is carried from a primary focus, or if two or more clumps fuse, the small vessel is closed and the foundation for a suppurative osteomyelitis is laid.

The yellow staphylococci are found more frequently than the white in these lesions, as the former occur more frequently in the primary lesions in the skin, mucous membrane, etc.

Hæmatogenous suppurative osteomyelitis may be caused:

1. By the deposition of highly virulent bacteria in the bone marrow.
2. By the lodgment of infected or bacterial emboli.
3. By the development or fusion of clumps of staphylococci in the finest capillaries.
4. The relation of trauma to osteomyelitis must also be considered.

The relation between trauma (in the broadest sense, fractures, contusion, and cold) and osteomyelitis is threefold:

1. Pyogenic bacteria which have gained access to the blood stream are apt to be deposited where the tissues are injured (locus minoris resistentiæ).
2. The trauma may injure the tissues in which bacteria have already been deposited, and so reduce their natural resistance that the bacteria may multiply and invade the tissues.
3. The trauma may rupture the connective tissue or bony capsule which surrounds some old focus.

The age at which the disease develops and the position of the focus differ. The greatest number of cases develop between the eighth and seventeenth years. The disease rarely develops after the twenty-fifth year. According to Haaga 59 per cent of the cases occur in the second decennium, 9 per cent in the third, 2.5 per cent in the fourth, and 2 per cent in the fifth.

Animal experiments coincide with clinical experience as to the age in which osteomyelitis is most frequent. While young animals after intravenous injections of staphylococci develop suppurating foci in bone and some of the other tissues, older animals develop a suppurative arthritis, never intraosseal, and only rarely periosteal foci (Rodet, Lexer).

This difference depends upon the greater vascularity of young growing bone and the histological characteristics of the cellular marrow of young bone which differ markedly from those of the fatty marrow of adult bone.

The more frequent occurrence of the disease in country people is probably due to the fact that they are less cleanly than city people (Kuester).

Osteomyelitis develops most frequently in the long hollow bones, and in that part of the bone in which the changes associated with growth are most active. According to different statistics the lower end of the femur, the upper end of the tibia, the upper end of the humerus, and the lower end of the tibia must be regarded as the favorite sites for the development of suppurating foci. They are involved in order of frequency as given above. In the epiphysis of long hollow bones, in the short and flat bones where tuberculosis develops frequently, suppurative osteomyelitis rarely occurs. Many different bones or different parts of the same bone may be involved simultaneously.

Clinical Forms.—Clinically suppurative osteomyelitis may be divided into acute and chronic forms with a number of complications. Classification based upon the sequelæ and bacterial forms may also be made.

A sudden onset and severe course are characteristic of ACUTE HÆMATOGENOUS SUPPURATIVE OSTEOMYELITIS. Strong, previously healthy children or young adults suddenly present the symptoms of severe infection (chills and high fever) and complain of a severe localized pain. If an extremity is involved the pain may be severe enough to prevent any movement. At first the patient may be unable to indicate accurately the location of the pain, but it soon becomes localized in a part of the bone, usually close to a large joint.

Often any external cause is wanting, often there is undoubted connection with a trauma; often a chronic suppuration, especially after a trauma becomes acute. In rare cases a suppurative osteomyelitis develops at the seat of a subcutaneous fracture, secondary to an angina, which developed during the process of repair.

The fever, accompanied by the severest general symptoms, is continuous. In the course of one or more days the affected extremity swells and presents slight indistinct redness, soon also inflammatory œdema, tension of the skin, fluctuation—in short, all the signs of a phlegmon of the soft tissues. The bone, if it can be palpated through the infiltrated tissues, appears to be thickened.

The subcutaneous veins are prominent, the neighboring lymphatic glands enlarged and sensitive to pressure. Abnormal mobility of the epiphysis and slight dislocation indicate separation of the epiphysis, which occurs in from twelve to fifteen per cent of the cases (Garré, Reisz). This occurs, as a rule, at the end of the first week, rarely as early as the second day. The neighboring joints often become involved in the inflammation (*vide* Complications).

A few days after the onset a serous infiltration of the soft tissues surrounding the bone, particularly the intermuscular septa, is found if an operation is performed. The discolored periosteum, which may be perforated at different points, is raised from the bone by pus, and the white bone is surrounded completely or partially by it. Where the periosteum retains its connection with the soft tissues, it is able to regenerate. Upon closer inspection one sees pus discharged from the large canals of the metaphysis, which contain vessels, and small drops of fat floating upon the surface of the pus, which indicate that the latter has been discharged from the medulla. Pus is found in the beginning only in that part of the medulla adjacent to the epiphyseal cartilage. The remaining marrow is deep red in color, and through it are scattered yellowish spots and streaks. Later it becomes transformed into a yellowish green collection of pus. Microscopically groups of cocci are found in the pus.

FIG. 107. — FOCI OF STAPHYLOCOCCI IN THE NECK OF THE FEMUR, INTRACAPSULAR RUPTURE (TWO YEAR OLD CHILD).

The bones more rarely involved are attacked in the severest multiple forms, which comprise about one fifth of the cases (Garré), in addition to the one usually affected. The bones become infected simultaneously from some primary focus or secondarily to some osteal focus (Garré). Sometimes the bones become involved simultaneously or in rapid succession, sometimes after long intervals. Sometimes these cases pursue an acute, at other times a chronic course. Multiple osteomyelitis, like hæmatogenous osteomyelitis, is, as a rule, a general metastatic infection with pyogenic micro-organisms. It occurs in children, whose bone marrow is especially susceptible to metastatic inflammation.

An acute hæmatogenous suppurative periostitis is most frequently associated with small suppurating foci in the cortex of flat bones and suppuration in the metaphysis of long, hollow bones. It occurs also in adults.

Those cases of acute osteomyelitis in which the focus develops in the articular ends of bone have been placed in a special group, because the joints are so frequently involved (W. Müller). In this form, which has been observed in the very young up to the fifth year, small suppurating foci exist. They are situated like the tuberculous foci, where the vessels from the metaphysis or periosteum enter the epiphyseal cartilage, or in the femur, where the vessels from the ligamentum teres, in the knee from the crucial ligaments, enter and branch (Fig. 106). Foci are found in the periosteum, cortex and medulla. The carpal and tarsal bone may also be involved (Becker). In the hip joint foci are found in the upper angle of the Y-shaped epiphyseal cartilage. Staphylococci

are found most frequently in this form of osteomyelitis, the streptococcus and pneumococcus relatively frequently.

Suppurative arthritis, or after extracapsular rupture para-articular phlegmon, are the most prominent clinical features. When the operation is performed a focus is found, which should be thoroughly removed with a sharp spoon to prevent destruction of the epiphysis and its cartilage and to protect the joint from subsequent inflammation.

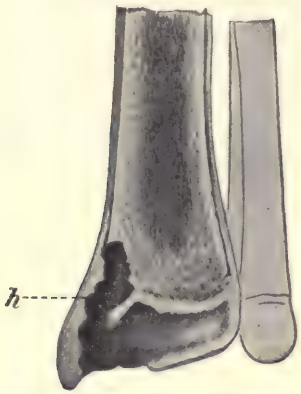

FIG. 108.—PNEUMOCOCCIC FOCUS IN THE INTERNAL MALLEOLUS, RUPTURE THROUGH THE EPIPHYSIS (NINE MONTHS OLD CHILD).

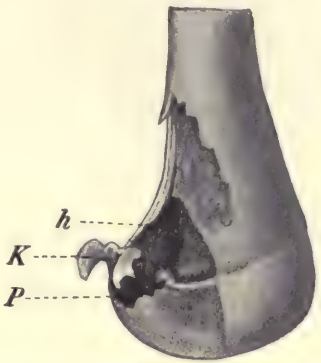

FIG. 109.—PNEUMOCOCCIC FOCUS IN LOWER ARTICULAR END OF THE FEMUR OF A CHILD NINE MONTHS OLD. *P*, Rupture through the epiphysis; *K*, capsule of joints.

OSTEOMYELITIS and PERIOSTITIS SEROSA (albuminosa of Ollier, non-purulenta of Schlange) is a rare form, which develops most frequently in the femur and is produced by the yellow and white staphylococcus and streptococcus. After a mild but acute onset it often pursues a chronic course and leads to the formation of large periosteal abscesses and suppurating foci in bone and sequestra. A serous or mucoid exudate is found instead of pus, and it differs in this way from the suppurative form. This form has been classified by Schlange with acute osteomyelitis because staphylococci have been demonstrated in the exudate. The inflammatory reaction is not so severe, however, as only a serous exudate is formed. According to Vollert and Garré it is possible that the pus, previously formed, may have undergone a mucoid degeneration. An exudate occurring in periostitis, which is surrounded by a thick resistant membrane, may resemble a cyst (periosteal ganglion, or if occurring on the skull, it may be mistaken for a meningocele, Schrank). The superficial necrosis of bone and the demonstration of pyogenic bacteria make certain the diagnosis.

Inflammation of the joints is the most important complication of acute suppurative osteomyelitis. The joints may become infected in

three ways. The rupture of a suppurating focus through the articular end, or of a medullary phlegmon through the epiphysis produces an acute suppurative arthritis. The arthritis may develop as a metastatic infection from a primary focus or from the bone primarily involved. It is then accompanied by a serous or purulent exudate. The third form of arthritis is the so-called sympathetic. A serous exudate, which apparently is produced by the toxins, develops, although the focus, acute or chronic, in the epiphysis is completely encapsulated. This form of arthritis may present the clinical features of an intermittent hydrops.

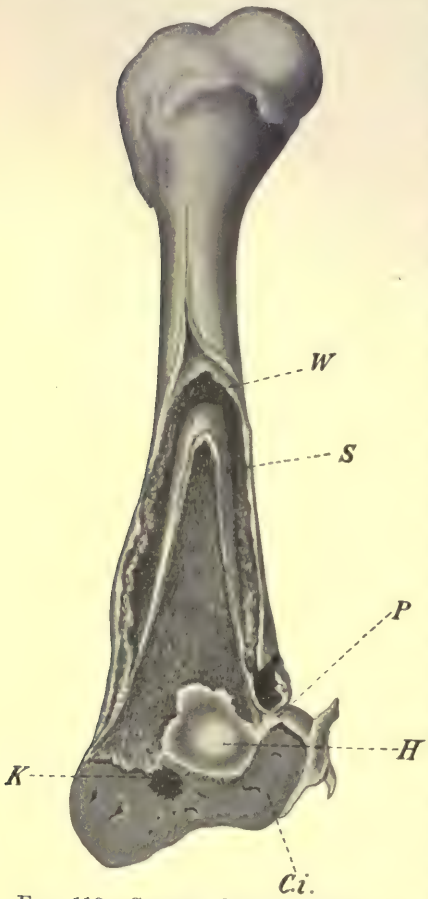

FIG. 110.—SEVERE OSTEOMYELITIS OF THE FEMUR IN A CHILD NINE WEEKS OLD, CAUSED BY STREPTOCOCCI; THREE WEEKS AFTER THE BEGINNING OF THE DISEASE. *K*, Center of ossification; *H*, suppurating focus; *Ci*, internal condyle; *P*, perforation; *W*, periosteal bone formation; *S*, sequestration.

When the exudate is large, the capsule may become greatly distended and subluxation occur.

Aspiration and immobilization of the joint, as a rule, control the arthritis, accompanied by the formation of large serous exudates. When, however, the exudate is purulent, the joint must be incised and drained immediately in order to retain good function. Resection of the joint must be considered when the epiphysis and articular cartilage are destroyed by the rupture of a medullary phlegmon (Fig. 111). Pathological changes in the articular cartilage produce adhesions and anchylosis (*vide* Diseases of Joints).

The most DANGEROUS COMPLICATION IS GENERAL INFECTION. If in a few days a fatal blood infection develops, in addition to the infection of one or more bones, it is impossible to say whether the general infection is secondary to the infection of the bone or whether the bone infection has occurred in the course of the general infection. In these cases no pus is found in the inflamed bones, but only hæmorrhagic foci scattered throughout a hyperæmic marrow (acute hæmorrhagic osteomyelitis).

Metastatic suppuration in the serous cavities and joints and purulent foci in the organs and muscles gives to this form of osteomyelitis the picture of a general metastatic infection. As in multiple osteomyelitis, the metastases may develop simultaneously from some primary focus or an osteal focus.

Other complications depend upon the position of the bones involved. An empyema may develop from an osteomyelitis of the bones of the thorax or vertebræ.

The severest cases, in which the symptoms of a general infection are most prominent, may end fatally within a week (the typhus of bone of French authors).

As a rule, the fever and general symptoms subside as soon as the bone is opened and drained or the pus is discharged spontaneously. The mild subacute cases subside spontaneously after a few days, as the infection is encapsulated. Acute osteomyelitis should always, however, be regarded as a grave disease. Metastatic and general infection and complications of all sorts threaten the life of the patient, the destruction of the diseased bone, and the function of the joint.

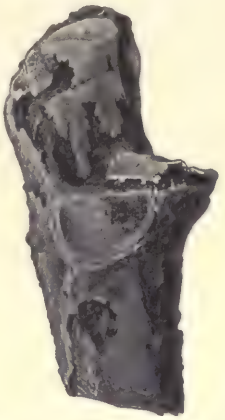

Fig. 111. — Suppurative Inflammation of the Elbow Joint Secondary to Osteomyelitis of the Ulna. Articular cartilage of the fossa semilunaris destroyed and fibrillated.

In the acute febrile stage and in the febrile relapses bacteria may be cultivated from the blood (Garré, Sänger, von Eiselberg, Canon, Lexer). The prognosis is bad, if the blood infection persists for some days after the focus has been opened and drained. Yet recovery has occurred in cases in which the bacteria have persisted in the blood for weeks (Lexer).

Diagnosis.—The acute violent onset with the symptoms of general infection and the symptoms of local inflammation are important in making the diagnosis of acute suppurative osteomyelitis. The diagnosis is not difficult if the local symptoms are found in bones, which are frequently involved, and if the inflammatory exudate in soft tissues can be traced to the bone and a direct wound infection or lymphangitic abscess can be excluded. Frequently a felon, a furuncle, an inflamed fissure or wound (e. g., scalp wound), an eczema, a scratch, a tonsillar abscess or an otitis media affords the infection atrium.

Osteomyelitis is most frequently confused with deep lymphangitis and lymphangitic abscess, especially if these develop in parts (popliteal fossa, Scarpa's triangle, and internal bicipital sulcus), which are frequently secondarily involved in osteomyelitis, and with large hæma-

togenous muscle abscesses. The incision which is necessary in the treatment makes the differential diagnosis possible. If the periosteum is firmly attached to the bone, the inflammation did not develop in the latter.

The treatment of acute suppurative osteomyelitis should protect the patient from general infection and limit the necrosis of the bone. The earlier the focus is opened, so much the better will both indications be met.

The incision should be made slowly, under general anæsthesia, through the intermuscular septa to the surface of the bone. If possible, artificial ischæmia should be employed, so that nerves, tendons, and blood vessels may be avoided. The yellowish discolored periosteum, raised from the bone, should be incised; the extent of the incision depending upon the extent of the suppuration. If the subperiosteal pus contains fat drops and is discharged from the bone the medullary cavity and the spongy tissue of the metaphysis should be opened. The compact bone should be removed by a chisel, and the entire suppurating focus exposed. The operator should avoid injuring the capsule of the joint, fracturing thin bone, and separating the loosened epiphysis. If the suppuration has extended to the epiphysis the articular cartilage should be spared. After the pus in the medulla and spongy bone has been removed by sponges or a sharp spoon the cavity in the bone and the wound should be tamponed with iodoform gauze.

The general rules already given should be followed in applying the dressing, which should hold the fragments in apposition if the epiphysis has been separated, and in the after-treatment, which will be required for from three to five months.

Frequently small sequestra are extruded while the bone cavity and the wound are closing by granulation tissue and the periosteum is forming new bone. Apparently after early operation the greater part of the remaining bone repairs and contributes to later growth. Incision of the abscess of the soft tissues without opening of the bone as well as drilling the latter at a number of different points is not enough. These methods of treatment do not provide for a free discharge of pus from the bone. They favor and cause chronic suppuration, extensive necrosis, rupture into the joint, acute relapses, etc.

The complete removal of the diseased part must be considered, if the epiphysis is necrotic, or if the shaft of a long bone is separated at both epiphyseal cartilages, is surrounded by pus, and no longer connected with living tissues. This occurs in the humerus, ulna, fibula, most frequently in the tibia and fibula of small children. In spite of such an extensive necrosis as above mentioned, the periosteum is able to form new bone, and is aided by isolated periosteal rests and the surrounding

connective tissue. It is necessary to resect the infiltrated parts in flat bones (ilium, scapula, ribs). In the skull bones it is often necessary to trephine, in addition to chiseling away the diploë, in order to provide drainage for subdural abscesses.

In the severest forms of osteomyelitis of the long hollow bones, amputation or disarticulation may be necessary in order to overcome the general infection. This is of advantage only when the infection in the bone is not localized.

Chronic suppurative osteomyelitis (osteomyelitis chronica purulenta) develops from the acute form and also occurs as an independent form. It follows open injuries of bone, periosteal suppuration, inflammatory processes about bone (e. g., varicose ulcers), and hæmatogenous infections.

If the acute stage of an osteomyelitis has subsided spontaneously after rupture and discharge of the pus, or if the pus has been discharged after incision of the soft tissues, the necrotic bone maintains an inflammation, which in long, hollow bones may persist for a half year or longer. This inflammation may separate or rarely digest the dead bone or produce osteoplastic changes. During all this time there is no trouble to speak of, unless the neighboring joint becomes inflamed or the epiphysis separates.

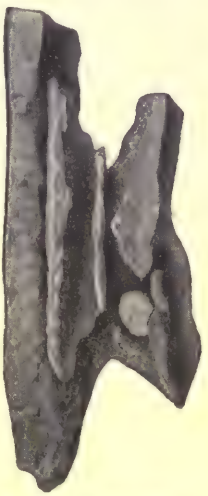

Fig. 112.—Central Sequestrum in the Lower Third of the Radius of Man Fifty Years of Age. The involucrum surrounding the dead bone is thick. Gradual enlargement of the bone for some years. Never acute inflammation and rupture. Few symptoms.

From time to time the fistula leading to the dead bone closes, and then the patient complains of throbbing pain in the bone, accompanied by fever, until the pus is discharged again. The bone is irregularly expanded, sometimes its entire circumference is involved, at other times only limited areas. When the patient comes to the physician an inflammatory infiltration with redness of the skin and deep fluctuation may be present. The opening of the fistula (if present) is surrounded by luxuriant granulations, and is not corroded, as in tuberculosis. When the pus is discharged small sequestra may be extruded. A larger, pointed sequestrum, which the patient attempts to remove, may be caught in the fistula. Neighboring vessels are sometimes injured in this way.

A severe, acute progressive suppuration of the bone, accompanied by a phlegmon of the soft tissues and high fever, follows, as a rule, an injury of the chronically inflamed bone. The cocci, which have re-

mained in the granulation tissue or scar for years without doing any harm, pass through the ruptured protecting capsule, invade the tissues again, and are absorbed.

The independent chronic forms have frequently a short, but not marked acute stage, which is often overlooked or forgotten. It occurs in the young as a febrile disease, associated with pain and swelling of one or more bones, which subside after a few days without the discharge of pus. After many years, even after full growth has been attained, pain develops in the area, which has always been somewhat expanded, but is now plainly thickened. This bony thickening, which may gradually become quite large, involves most frequently the ends (junction of metaphysis and epiphysis) of long bones, and not infrequently is accompanied by arthritis (suppurative synovitis, after rupture of an osteal focus, or intermittent hydrops) and by abscess formation in the soft tissues.

Three principal forms which frequently pass over into each other may be differentiated:

1. The central sequestrum surrounded by a very thick involucrum with little or no suppuration (Fig. 112).

2. The bone abscess, which is found most frequently in the metaphysis. It varies in size from a pea to a hen's egg, is lined by a thick abscess membrane, and contains thick, sclerotic bone. The bacteria (both varieties of the staphylococcus) found in these abscesses may remain viable for twenty or thirty years.

3. The sclerotizing osteomyelitis (Garré), which has an acute or subacute onset, but does not lead to pus formation, is to be regarded as a less active form. The at times painful, gradually thickening bone, is transformed finally into a solid mass, which, as in syphilitic hyperostoses, may encroach upon the marrow cavity. A large area or only the ends of the bone may be transformed into such a mass. Very small abscesses, foci of granulation tissue, and small central sequestra are frequently found within this sclerotic bony tissue.

Acute exacerbations, which may follow trauma and other diseases, are the dangers of chronic suppurative osteomyelitis. Abscesses, inflammation and disturbance of the function of joints and different sequelæ may develop even after long intervals.

The diagnosis of the chronic form may be difficult if other signs do not indicate the nature of the changes, which may not be very distinct even in the Roentgen ray picture. The diagnosis of sarcoma, tuberculosis, gumma, and bone cyst may be made, therefore in doubtful cases an exploratory incision should be made.

The swelling which develops insidiously upon the ends of bones, near joints, and upon short bones, such as the clavicle, without the

signs of inflammation and fistula formation, resembles myelogenous or periosteal sarcomas. At first they develop slowly, after a time more rapidly, and produce pain, functional and circulatory disturbances. The development of an inflammatory infiltration and reddening of the skin are the surest signs of the inflammatory nature of this chronic process. In rare cases (Kocher, Jordan) the swelling is composed mostly of granulation tissue, and the compact bone covering it is thinned, so that a microscopic or bacteriologic examination must be made before it is possible to diagnose the nature of the swelling. Thick masses of periosteal scar tissue, resulting from previous inflammation, may lead to the diagnosis of sarcoma (Nasse, W. Mueller). When such a mass is exposed, small granulating and suppurating foci and sequestra will be found. Chronic epiphyseal foci, especially if they have produced a serous synovitis, may be mistaken for tuberculosis. The fistulæ following chronic osteomyelitis do not have the corroded borders which characterize the tuberculous. The pus in the small osteal foci is thick and mucoid, not caseous, the sequestra jagged and irregular, not round, as in tuberculosis. The swellings of the diaphysis, which occur in the non-suppurative sclerotizing form, and are characterized by frequently recurring bone pain and a chronic course, remind one of bone syphilis, especially if there is no acute stage.

Bone cysts resemble serous abscesses, which are encapsulated by thick connective tissue. The demonstration of bacteria makes a differential diagnosis possible, if it cannot be made from other data.

The treatment of chronic suppurative osteomyelitis consists in the removal of the sequestrum, the exposure and evacuation of the suppurating focus. If the focus is centrally situated the bone must be chiseled away (necrotomy). One may figure upon a complete separation of even large sequestra and the development of a strong involucrum if six months have passed since the beginning of the disease.

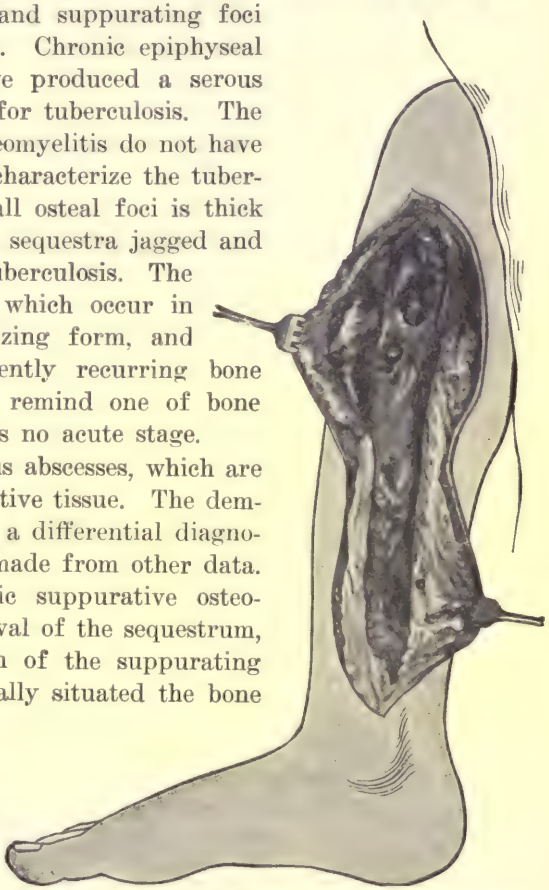

FIG. 113.—NECROSIS OF THE TIBIA WITH NUMEROUS CLOACÆ EXPOSED FOR SEQUESTROTOMY.

In exposing the focus the periosteum is incised the length of the swelling and reflected to either side. The exposed wall of bone is then

removed with a chisel or gouge. When the sequestrum is removed the granulation tissue, pus, and abscess membrane are removed by sponges or the sharp spoon. The sharp borders of the bone are cut away with a straight chisel, and the cavity in the bone is then tamponed. Foci and sequestra about the epiphyseal cartilage must be followed if necessary into the epiphysis. The epiphyseal and articular cartilages and joint capsule should not be injured.

Repair by the formation of granulation tissue is slow. Deep fistulæ, which extend into the metaphysis, and which must be curetted frequently, often remain.

The deep bone cavities near the articular ends, which cannot be smoothed off completely, because so near the joint, may be closed most easily in the following way: After healthy granulation tissue has developed, a pedunculated skin flap is made and placed upon the vivified granulating surface, or during the operation the skin flaps are so fashioned that after the removal of the tampon they may be turned into the wound. They may be held in position by dressings, small nails, or adhesive plaster.

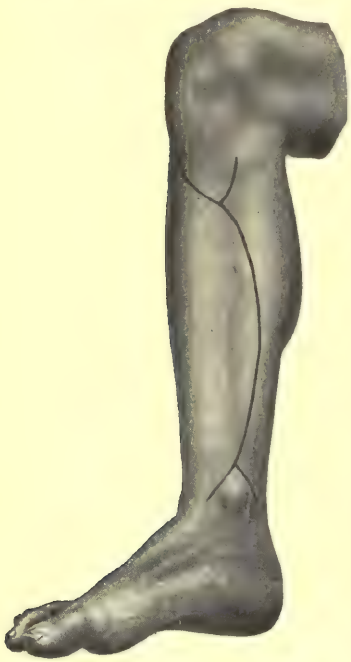

FIG. 114.—INCISION FOR EXPOSURE AND PARTIAL REMOVAL OF THE TIBIA IN EXTENSIVE SUPPURATIVE OSTEOMYELITIS.

Osteoplastic necrotomy (Luecke, Ollier, Bier), in which a piece of the involucrum, retaining a periosteal attachment, is used to fill in the cavity, has the disadvantage that dead spaces form beneath the flap and pus is retained. The process of repair is not shortened.

A number of attempts have been made to close these cavities with different kinds of plugs. The iodoform bone plug introduced by Mosetig-Moorhof, consisting of 60 parts of iodoform and 40 parts each of spermaceti and oil of sesame, has been the most successful. It does not act as a foreign body as other bone plugs do, and does not produce suppuration. It is gradually absorbed and replaced by connective tissue or newly formed bone, after the skin, which was immediately sutured, has been healed for some time.

The most important sequelæ are: Spontaneous fracture or infraction at the point where the bone has been weakened by the inflammatory process. The fracture occurs most frequently in the demarcation zone, where

the involucrum is poorly developed or it is weakened by an operation, in rare cases also at the site of the sequestrum, which is not sufficiently supported by the involucrum. The fracture may follow extensive suppuration. Necrotomy should be performed, and the fragments approximated and retained in as good position as possible. The repair of such a fracture is slow, pseudarthrosis often cannot be prevented.

Pathological dislocation occurs most frequently at the hip joint; subluxation at the knee joint. They result from the destruction of the joint (destruction-dislocation) or from distention of the capsule by effusions into the joints (distention-dislocation). Separation of the rim of the acetabulum, which then moves upon the ilium, may lead to the diagnosis of pathological dislocation.

Bending may occur at the weak point of the involucrum if weight is borne upon the leg or as the result of muscular contraction, e. g., this bending may be forward in the lower end of the femur, displacement backward of the upper end of the tibia from contracture of the flexor tendons. The most marked deformities follow separation of the epiphysis, with subsequent imperfect repair. These deformities are also partly due to irregular growths resulting from disease and destruction of the epiphyseal cartilage.

FIG. 115.—RADIOFLEXION OF THE HAND FOLLOWING DESTRUCTION OF THE LOWER EPIPHYSIS OF THE RADIUS BY A SUPPURATIVE OSTEOMYELITIS.

Disturbances of growth consist of shortening and lengthening of the diseased and neighboring bones (Ollier, von Bergmann, Helferich). Shortening of the bone follows the destruction of the epiphyseal cartilage, which does not regenerate. Foci in the diaphysis and metaphysis may stimulate the zone in which growth is most active, and produce a lengthening of the bone. Either of these changes, depending upon the

position of the focus, may be produced experimentally (Lexer). These pathological changes occurring in the bones of the forearm and leg may give rise to a number of deformities (pes valgus, varus, manus radioflexa, genu valgum, varum, etc.), for the plane of the joint is displaced and deformities of the rapidly growing bones (healthy or diseased) result. Moreover, the healthy bones of an extremity may be increased in length as the result of the increased blood supply accompanying the inflammation. In inflammation of the bones of the leg, the femur may increase in length and the reverse; in osteomyelitis of the bones of the forearm the humerus may become longer. In this way the shortening of the diseased bone is compensated. According to Ollier, when a bone of an extremity becomes shortened, there is a compensatory lengthening of the bone adjacent to it.

Fig. 116.—Marked Curvature of the Tibia Resulting from Shortening of the Diseased Fibula.

Contractures with fibrinous adhesions and anchylosis may follow the inflammation of the joints occurring in the course of an osteomyelitis.

Von Volkmann has designated as recurrent osteomyelitis the form which develops after an interval of years upon a completely healed osteomyelitis. The old area may be involved, or a bone which has been perfectly healthy. There is either the invasion by bacteria, which have remained latent about the old focus, or there is a new blood infection. It is possible to explain in this way the development of inflammatory changes in old osteomyelitic foci and in healthy bone. The scar tissue of the old focus may contain latent bacteria, or it may be the locus minoris resistentiæ, where the bacteria circulating in the blood are deposited. When the inflammation occurs in bone not previously involved, one cannot exclude a focus which developed without symptoms in youth and remained latent.

Bacteriology.—A classification of hæmatogenous suppurative osteomyelitis based upon the bacterial forms cannot, as a rule, be made. The clinical differences between the inflammations produced by the different bacteria are not striking enough to make this possible.

Streptococci produce, according to our present knowledge, small cortical and metaphyseal foci with suppurative arthritis (especially in children) as well as medullary phlegmons with separation of the epiphysis and extensive necrosis. Streptococci also produce osteitis albuminosa,

abscess, chronic thickening, and inflammation of the flat bones. The pus is thin, milky, discolored green, and is formed in large quantities. Mixed infections of staphylococci and streptococci produce severe local and general symptoms.

Pneumococcic osteomyelitis is more rare. The foci are situated in the ends of the bone near the joints, from which they produce suppurative arthritis. The pus resembles the streptococcic pus. There develops in rare cases in children and adults during the course of pneumonia periosteal and cortical suppuration, and also suppurative inflammation of subcutaneous fractures (Lexer).

A gonococcic osteomyelitis (in the humerus of an adult) has been observed once by Ullmann. A perichondritis of a rib has been observed by Finger.

The bacterium coli commune has been found in some cases (Klemm, Mauclaire) associated with the typhoid bacillus and staphylococcus. Its presence is indicated by foul-smelling and discolored pus.

Schlangenhaufer found the bacillus of pneumonia (Friedlaender) in an extensive osteomyelitis in an adult. There are also rare cases of actinomycosis in which the fungus has been found in an osteal focus (Wrede, Fig. 142, p. 369). Wyss found an anærobic bacillus (Bacillus halo septicus) in the ichorous pus of an osteomyelitis of the tibia.

Typhoid osteomyelitis (osteomyelitis typhosa) developing in the course of or subsequent to typhoid fever demands a separate consideration. It develops, as a rule, in from the fourth to the sixth week of the disease; sometimes after many years. It is caused by the typhoid bacillus, frequently associated with the ordinary pyogenic bacteria, which pass from the intestinal ulcers into the blood and are finally deposited in the bones. Its onset is indicated by a rise of temperature and pain in the bone involved. Trauma is frequently the predisposing cause. The resulting abscess, which is often very large, contains, if there is no secondary infection, a yellowish-brown, rust-colored fluid, the so-called typhoid pus, which may be sterile. It does not differ from ordinary pus if secondary infection with the pyogenic cocci occurs.

Fig. 117.—Typhoid Focus in a Costal Cartilage.

When the abscess ruptures, external suppurating fistulæ, which are maintained by small granulating and necrotic foci, are produced. These fistulæ, which are very resistant to treatment, remind one of tuberculous fistulæ.

Typhoid osteomyelitis develops most frequently in the ribs. The foci are situated in the costal cartilages close to their articulation with the ribs or in the latter, in which are found small total sequestra sur-

rounded by a thick granulation tissue and a thin involucrum. Cortical and central foci develop in the tibia; the involvement of other bones (pelvic and skull bones, clavicle, sternum, humerus, femur, vertebræ, spondylitis typhosa, Quincke) is rare. The osteomyelitis has an acute stage, and then pursues a chronic course. It is not rare for multiple foci to develop. The inflammation remains localized and there is but little reactive bony growth. If the disease resembles acute suppurative osteomyelitis there is either a mixed infection or infection with the staphylococcus or streptococcus alone (*vide* Secondary Osteomyelitis).

Ebermaier and Quincke made the important observation that typhoid bacilli are found as regularly, and almost in as large number, in the red bone marrow as in the spleen of patients dying of typhoid fever. They remain viable for a long time, as they have been found in osteal foci six to seven years after convalescence from typhoid fever (Sultan, Buschke, and others). According to Ponfick these bacteria produce slight but general alterations in the bony system, partly periosteal thickening, partly superficial caries. These findings make clear the relation existing between trauma and typhoid osteomyelitis. Pyogenic cocci may also pass through the diseased intestinal mucous membrane, enter the blood, and cause inflammation of bone even in adults, for the resistance of the bone marrow has been reduced by the typhoid infection.

Suppurative inflammation of bone following infectious diseases is called secondary osteomyelitis.

Before such an osteomyelitis develops, there must be a localization of the specific organisms in bone. In the metapneumonic osteomyelitis pneumococci and streptococci are found. Infection atria are provided in the course of infectious diseases for the ordinary pyogenic bacteria, and the resistance of the bone marrow is also probably reduced, and is therefore more susceptible to infection. This form of osteomyelitis develops in the course of or subsequent to typhoid fever, pneumonia, measles, scarlet fever, diphtheria, influenza, and smallpox.

Phosphorus necrosis is also a secondary but not a pure suppurative osteomyelitis. Chronic phosphorus poisoning, which follows the inhalation of phosphorus fumes in the preparation of crystalline phosphorus and the manufacture of phosphorus matches, is associated with changes in the bones.

Some post-mortem examinations have demonstrated an ossifying process (phosphorus periostitis and sclerosis), while clinical observations have demonstrated abnormal softness and fragility of the bones. Some patients have had multiple fractures (a case reported by Haeckel suffered thirteen fractures in sixteen years). The nature of these changes, especially those occurring in the jaws, is not exactly known. There

appears to be some direct relation to the phosphorus fumes which are inhaled.

Continued feeding of small amounts of phosphorus produces in young animals an osteosclerosis (Wegner). On the other hand, a direct action upon exposed bony surfaces cannot be demonstrated (von Stubenrauch).

FIG. 118.—PHOSPHORUS NECROSIS OF THE MANDIBLE AFTER HAECKEL. Removed from a woman twenty-five years of age. At *k*, beginning line of demarcation, at *l* a cloaca in the depths of which a cortical sequestrum may be seen. Osteophytes cover the surface of the bone.

The most important changes occur in the mandible and maxilla, especially in people who are engaged in the manufacture of matches. These changes were first described by Lorinser in 1845.

The first changes, which sometimes consist of greater fragility of the jawbones, sometimes of thickening and sclerosis, develop without symptoms and insidiously. The symptoms of the disease, which is to be regarded as a secondary suppurative or sanious osteomyelitis, developing in bone already altered by phosphorus fumes, begin with an inflammation of the gums and periosteum. This inflammation is caused by the different pyogenic and putrefactive bacteria of the mouth cavity which gain access from carious teeth, small ulcers, and injuries to the gums and periosteum. Some of the teeth become loosened, yet there is no improvement, as in the ordinary periostitis alveolaris, when they are extracted. Pain and swelling increase, the floor of the mouth and the

cheeks become infiltrated. Later suppuration occurs, and fistulæ are formed from which is discharged foul-smelling pus. Other teeth become loosened, the gums and the periosteum are raised from the bone by a layer of pus, and the surface of the bone is exposed. A large part of the bone involved may be destroyed by the chronic progressive inflammation; the entire mandible may become necrotic in from six to nine months. The remaining healthy periosteum forms a thick involucrum. The sequestrum, the edges of which become osteoporotic, separates slowly. In extensive necrosis two to three years are required for the separation of the sequestrum. Healing occurs after the sequestrum is extracted. In the meantime anchylosis of the jaw, disturbances of digestion, due to the swallowing of pus, poor nutrition, pain in the entire jaw, and other symptoms which are frequently accompanied by fever develop. The patient becomes weaker, and complications such as meningitis, general infection, and pneumonia may prove fatal. Death occurs in about one half of the cases.

The mandible is involved about nine times more frequently than the maxilla, and the necrosis occurring in the former is much more extensive.

The treatment is prophylactic and operative. The workrooms in match factories should be well ventilated; the mouth hygiene should be good; employees should not be allowed to eat or drink in the workroom; and the hands should be carefully washed after work. The teeth of the employees should be inspected frequently by a dentist, and no person employed who has bad teeth. By proper ventilation of the factory and proper care of the teeth of the employees, the largest match company in America, the Diamond Match Co., has practically eliminated the disease. The operative treatment consists of early and extensive subperiosteal resection of the diseased bone.

After the periosteum and the osteophytes attached to it are separated, all the diseased bone is removed, the resection being carried into healthy tissues. Small partial resections of the alveolar process should not be made, but the middle piece, a half of the mandible, or the entire bone should be removed, depending upon the extent of the pathological changes, and the same treatment should be employed in necrosis of the maxilla (Riedel, Haeckel). Suppuration ceases and repair follows after complete removal of the diseased bone. The periosteum regenerates bone so rapidly that good functional and cosmetic results are soon obtained, even after a total resection.

Literature.—*A. Becker* (W. Müller). Ueber einen ungewöhnlichen Ausgang der akuten Osteomyelitis. Deutsche Zeitschr. f. Chir., Bd. 55, 1900, p. 577.—*v. Bergmann.* Ueber die pathol. Längszunahme der Knochen. Petersb. med. Zeitschr., Bd. 20.—*Braasch.* Ueber pathol. Wachstum der Extremitätenknochen im Gefolge akuter Osteomyelitis. I.-D. Berlin, 1897.—*Dmochowski* und *Janowski.* Ueber Eiterung

erregende Wirkung des Typhusbazillus. Zieglers Beitr. z. path. Anat., Bd. 17, 1895, p. 221.—*Enderlen.* Histol. Untersuchungen bei experim. erzeugter Osteomyelitis. Deutsche Zeitschr. f. Chir., Bd. 52, 1899, p. 293.—*Franke.* Ueber einige chir. wichtige Komplikationen und Nachkrankheiten der Influenza. Chir.-Kongr. Verhandl., 1899, II, p. 490.—*Funke.* Beitr. zur Kenntnis der akuten Osteomyelitis. Arch. f. klin. Chir., Bd. 50, 1895, p. 462.—*Gangolphe.* Maladies des os, Paris, 1894.—*Garré.* Ueber besondere Formen und Folgezustände der akuten inf. Osteomyelitis. Beitr. z. klin. Chir., Bd. 10, 1893, p. 241.—*Gelinsky.* Eine Skelettdurchleuchtung bei einem Fall von Pyämie. Fortschr. auf d. Geb. d. Röntgenstrahlen, Bd. 9.—*Haeckel.* Die Phosphornekrose. Arch. f. klin. Chir., Bd. 39, 1889, p. 555.—*Helferich.* Ueber die nach Nekrose an der Diaphyse der langen Extremitätenknochen auftretenden Störungen im Längenwachstum derselben. Deutsche Zeitschr. f. Chir., Bd. 10, 1878, p. 324.—*Hödlmoser.* Typhöse Erkrankungen der Knochen und Gelenke. Sammelref. Zentralbl. f. Grenzgebiete, 1901, p. 417.—*Jordan.* Die akute Osteomyelitis. Beitr. z. klin. Chir., Bd. 10, 1893, p. 587;—Ueber atypische Formen der akuten Osteomyelitis. Ibid., Bd. 15, 1896, p. 457.—*Kocher* und *Tavel.* Chirurgische Infektionskrankheiten, 1895.—*Küster.* Ueber Frühoperationen bei Osteomyelitis. Chir.-Kongr. Verhandl., 1894, II, p. 397.—*Lexer.* 1. Zur experim. Erzeugung osteomyelitischer Herde. Arch. f. klin. Chir., Bd. 48, 1894, p. 181; 2. Osteomyelitisexperimente mit einem spontan beim Kaninchen vorkommenden Eitererreger. Ibid., Bd. 52, 1896, p. 576; 3. Experimente über Osteomyelitis. Ibid., Bd. 53, 1896, p. 266; 4. Die Aetiologie und die Mikroorganismen der akuten Osteomyelitis. v. Volkmanns Samml. klin. Vortr., N. F., 173, 1897; 5. Zur Kenntnis der Streptokokken- und Pneumokokkenosteomyelitis. Arch. f. klin. Chir., Bd. 57, 1898, p. 879; 6. Die Entstehung entzündlicher Knochenherde u. ihre Beziehung zu den Arterienverzweigungen der Knochen. Ibid., Bd. 71, 1903, p. 1; 7. Weitere Untersuchungen über Knochenarterien u. ihre Bedeutung f. Krankh. Vorgänge. Ibid., Bd. 73, 1904, p. 481; 8. Untersuchungen über Knochenarterien u. s. w. Berlin, Hirschwald, 1904.—*v. Mangoldt.* Zur Behandlung der Knochenhöhlen in der Tibia. Arch. f. klin. Chir., Bd. 69, 1903, p. 82.—*v. Mosetig-Moorhof.* Die Jodoformknochenplombe. Zentralbl. f. Chir., 1903, p. 433;—Erfahrungen mit der Jodoformknochenplombe. Deutsche Zeitschr. f. Chir., Bd. 71, 1904, p. 419.—*Nasse.* Chirurgische Krankheiten der unteren Extremitäten. Deutsche Chirurgie.—*Ollier.* Traité expérimental et clinique de la régéneration des os et de la production artificielle du tissue osseux. Paris, 1867.—*Perez.* Die Influenza in chirurgischer Beziehung. Deutsche Zeitschr. f. Chir., Bd. 63, 1902, p. 460.—*Reiss.* Klinische Beobachtungen über Osteomyelitis der langen Röhrenknochen, besonders in Bezug auf die Epiphysenknorpelfuge und die begleitenden Gelenkaffektionen. Arbeiten aus v. Bergmanns Klinik Berlin, Bd. 15, 1901.—*Regnault.* De la longeur relative des os. Bull. et mém. de la société anatom. de Paris, 1900, No. 5.—*Riedel.* Ueber Phosphornekrose. Chir.-Kongr. Verhandl., 1896, II, p. 485.—*Rieffel* et *Mauclaire.* Maladies des os. Traité de chirurgie, le Dentu et Delbet, Paris, 1896.—*Roeseler.* Beitr. zur Osteomyelitis mit besonderer Berücksichtigung der Therapie und der Heilerfolge. v. Volkmanns Samml. klin. Vortr., N. F., 243.—*Schanz.* Ueber Spondylitis typhosa. Arch. f. klin. Chir., Bd. 61, 1900, p. 103.—*Schlagenhaufer.* Osteomyelitis durch Bacillus pneumoniae. Zentralbl. für Bakteriol., Bd. 31, 1902, p. 73.—*Schlange.* Ueber einige seltenere Knochenaffektionen. Arch. f. klin. Chir., Bd. 36, 1887, p. 97.—*M. B. Schmidt.* Akute eiterige Osteomyelitis. Ergebn. d. allg. Path. Lubarsch-Ostertag, January 5, Wiesbaden, 1900, p. 956.—*Schrank.* Ueber einen Fall von seröser Osteomyelitis am Hinterhaupte, der eine Meningocele vortäuschte. Berl. klin. Wochenschr., 1902, p. 780.—*Schuchardt.* Die Krankheiten der Knochen u. Gelenke. Deutsche Chir., 1899.—*Silbermark.* Ueber die gewebl. Veränderungen nach Plombierung von Knochenhöhlen. Deutsche Zeitschr. f. Chir., Bd. 75, 1904, p. 290.—*v. Stubenrauch.* Die Lehre von der Phosphornekrose.

v. Volkmanns Samml. klin. Vortr., N. F., 303.—*Trendel.* Beitr. z. Kenntnis der akut. infekt. Osteomyelitis. Beitr. z. klin. Chir., Bd. 41, 1904, p. 607.—*Ullmann.* Osteomyelitis gonorrhoica. Wien. med. Presse, 1900.—*v. Volkmann.* Die Krankheiten der Bewegungsorgane, 1865.—*Vollert.* Ueber die sogen. Periostitis albuminosa. v. Volkmanns Samml. klin. Vortr., 352, 1890.—*Wegner.* Der Einfluss des Phosphors auf den Organismus. Virchows Arch., Bd. 55, 1872, p. 11.—*Weichselbaum.* Veränderungen der Knochen bei den akuten Infektionskrankheiten. Verhandl. der Gesellsch. deutscher Naturforscher, 1894, Wien.—*Wrede.* Hämatogene Osteomyelitis durch Aktinomyces. Chir.-Kongr. Verhandl., 1906.—*Wyss.* Ueber einen neuen anaërob. path. Bac. Mitteil. a. d. Grenzgeb., Bd. 1904, p. 199.

(f) PYOGENIC INFECTIONS OF JOINTS

Etiology.—Primary infection of a joint follows gunshot, contused and punctured wounds, the penetration of foreign bodies (needle, nail, pieces of glass or steel), compound dislocations and fractures. Secondary infection occurs when an adjacent phlegmon, erysipelas, acute and chronic suppurative osteomyelitis extends to a joint, or when a fistula, resulting from previous disease of the joint (e. g., tuberculous fistula), becomes infected.

Bacteriology.—Hæmatogenous arthritis develops when bacteria are deposited in the capillaries of the synovial membrane. This form of arthritis develops, as a rule, during the course of other infections, and an injury may be the predisposing cause. A multiple serous arthritis may develop in the course of an endocarditis caused by pyogenic bacteria, which may resemble clinically and be confused with acute articular rheumatism. Suppurative arthritis accompanies especially acute suppurative osteomyelitis and the general pyogenic infections. Serous and suppurative arthritis occurs in the course of a number of infections which afford infection atria for the ordinary pyogenic cocci (diphtheria, scarlet fever, measles, smallpox), or for the specific micro-organisms of the disease alone or combined with other bacteria (typhoid fever, pneumonia, gonorrhea, erysipelas, epidemic cerebrospinal meningitis, influenza).

Not only staphylococci and streptococci, but also the rarer forms of pyogenic bacteria, among these the bacillus of pneumonia and the meningococcus, are found in the different forms of arthritis. The staphylococci and streptococci produce particularly the severe, but are also found in the mild forms.

Morbid Anatomy.—When one comes to the consideration of inflammation, the synovial membrane is the most important part of the joint. The lining of the joint capsule, the stratum synoviale, which differs from the stratum fibrosum external to it, extends to the edges of the articular cartilages. The free surface of the stratum synoviale is not covered with epithelium or endothelium, but by a thin layer of regularly arranged,

epithelial-like connective tissue, which is provided with fine, threadlike or larger leaflike processes, the synovial villi, some of which contain fat. The synovial membrane and its villi are very vascular, and the capillaries penetrate into the fine, epithelial-like connective tissue.

The synovial membrane is also provided with a well-developed lymphatic plexus, which is not, however, in open communication with the cavity of the joint, as the lymphatic plexuses of the serous membranes are with the serous cavities.

Clinical Forms.—The symptoms of inflammation follow infection. A small amount of the exudate is poured out into the tissue of the capsule, the greater amount into the cavity of the joint. It makes no difference whether the bacteria have been carried into the loose connective tissues of the synovial membrane by injury, have reached it through the blood, or whether an osteomyelitic focus has ruptured into the joint and infected the entire surface of the membrane. We distinguish according to the character of the exudate three principal forms of synovitis (arthritis if not only the synovial membrane, but all the tissues of the joint are involved), the serous, fibrinous, and suppurative. There are a number of transitional forms. Sometimes a fourth form, the ichorus synovitis (in open wounds), occurs when there is an infection with putrefactive bacteria.

Serous and serofibrinous synovitis is a mild form. It develops after open injuries of the joints, secondary to encapsulated suppurating foci in the epiphysis, and to adjacent inflammation, in infectious diseases (pneumonia, typhoid fever, gonorrhea, etc.), and especially in general infections.

It develops acutely in one or more joints. The joint involved is painful, tense, and becomes considerably swollen. The skin covering it may be hot and reddened. The function of the joint is interfered with, there is some fever, and the general symptoms vary.

If there is a large amount of serous exudate, the joint capsule and the bursæ communicating with the joint become distended and prominent where anatomical relations permit (in the knee joint, at the sides of the ligamentum patellæ and upper recess). Fluctuation is plainly made out, and the patella is raised from its normal position and floats. The normal contour of the joint is lost, and is replaced by that of the distended joint capsule (*vide* Tuberculous Hydrops).

If, on the other hand, there is but a small amount of exudate, but considerable infiltration of the capsule and fibrinous masses are deposited upon the synovial membrane, tumorlike thickenings may be felt, especially at the points of reflection of the capsule, which often creak when palpated or moved. These thickenings, together with an œdematous infiltration of the peri- and para-articular tissues, render the outlines of the

joint indistinct (particularly in the phlegmonous form of gonorrheal arthritis).

As a rule, when serous synovitis is properly treated, the serous exudate is absorbed and the inflammation subsides without leaving any articular changes. If the inflammation recurs, a chronic condition with hydrarthrosis and growth of villi, as in traumatic arthritis, may develop. Then, without any pathological changes in the articular cartilages or bones, the distended capsule may permit of abnormal movements (flail joint) or the development of luxations and subluxations (for example, in typhoid fever, scarlet fever, and smallpox).

If the tissues of the capsule become inflamed, they may shrink and produce permanent disturbances of motion. These will be still greater if there has been a large fibrinous exudate which produces adhesions (especially in gonorrheal arthritis).

The serous exudate, a yellowish fluid somewhat clouded by pus corpuscles, contains less mucin, but more albumin, than synovial fluid.

The fibrinous exudate contains, besides small or large amounts of serous exudate, large amounts of fibrin, which occurs in acute infections in the form of flakes or membranes, loosely attached to the recesses, folds, and villi of the synovial membrane. In chronic inflammations these masses of fibrin become firmly attached to the hypertrophied synovial membrane and produce fibrinous, or if organized, fibrous, adhesions of the opposed surfaces.

Treatment of Serous and Serofibrinous Synovitis.—In the acute cases immobilization of the joint, after the removal by puncture of the larger exudates, is often sufficient. In the recurrent and chronic forms, aspiration combined with irrigation with from one to two per cent carbolic or boric acid solution is to be recommended. Early massage and careful passive motion are required in those cases in which there is a tendency to stiffness.

Besides, one may attempt to hasten the absorption of the inflammatory infiltration of the capsule by an artificially induced hyperæmia (painting with tincture of iodin, treatment with hot-air apparatus, Bier's passive hyperæmia).

Suppurative Synovitis.—Suppurative inflammation of joints (empyema) may be superficial or deep, and involve the synovial membrane or all the structures of the joint (synovitis and arthritis (para-arthritis) acuta purulenta).

The superficial suppurative joint inflammation (synovitis purulenta)—the catarrhal suppurative inflammation of joints of von Volkmann—is the mild form. The inflamed, reddened, and thickened folds of the synovial membrane secrete a profuse mucopurulent exudate which often contains fibrin flakes. The inflammation involves only the inner layers of

the capsule, and if proper treatment is instituted early there may be a restitutio ad integrum. If it persists for a long time and is neglected, this form of synovitis becomes a severe arthritis accompanied by destruction of the joint.

It accompanies, more frequently than the serous and serofibrinous forms, acute pyogenic infections. This form of synovitis is caused most frequently by staphylococci and streptococci; less frequently by pneumococci. Streptococci and pneumococci are found relatively frequently in the catarrhal suppurative inflammations of the joints, occurring in small children, and now and then in inflammations associated with small osteal foci (*vide* Osteomyelitis purulenta).

The *diagnosis* of suppurative synovitis is not difficult. It has an acute febrile onset and the local symptoms are marked. The joint is swollen and its outlines are rendered indistinct by the inflammatory œdema; there is loss of function, and severe pain is produced by palpation and movement. Nonsuppurative gonorrheal arthritis is the only form which resembles it.

The *treatment* consists of early and wide incision of the joint with the application of a loose tampon, later drainage of the wound and immobilization of the joint by splints. In many cases, especially in streptococcic and pneumococcic inflammation, aspiration of the pus is sufficient. If a suppurating osteal focus is the cause, it must be removed and the joint opened and drained. If all the inflammatory symptoms have subsided, careful active and passive motion should be employed. If begun too early the local condition may be aggravated or general infection produced.

Deep suppurative arthritis (arthritis purulenta) is not limited to the synovial membrane. It involves the peri- and para-articular tissues, or ruptures into the surrounding tissues and phlegmons, and gravitation abscesses develop in the spaces between muscles and fasciæ. Finally this form of inflammation destroys the capsule of the joint and the articular cartilages. The severest forms are caused by the staphylococci and streptococci.

It follows injuries, the rupture of acute or chronic osteal foci through the articular surfaces, or occurs as a metastatic inflammation in the course of general pyogenic infections.

The *diagnosis* is based upon the ordinary symptoms of arthritis and the two following characteristics: Phlegmonous inflammation of the tissues surrounding the joint indicating a rupture of the capsule; lateral mobility in the joint, associated with crepitation or subluxation, extensive destruction of the capsule, the ligaments, and articular cartilages.

The *treatment* should provide for the free discharge of pus from the joint and para-articular abscesses. Wide incisions should be made into

the soft tissues and free drainage established. In severe cases the incisions used for resections may be required. If the local and general symptoms do not subside and the destruction of the articular cartilages becomes more extensive, resection of the joint is indicated. In this way the entire inflammatory focus with all its pockets is completely exposed. Amputation is necessary only in the severest cases with extensive phlegmons, secondary involvement of the bone, and general infection.

Stiffening of the joints, deformities, and trophic disturbances are the most important sequelæ of the inflammation of joints. Stiffness of the joints may be caused by cicatricial contraction of the capsule, fibrous adhesions, and bony union of the articular surfaces. Interference with motion, due to cicatricial contraction of the capsule, may develop in the course of any infection which is accompanied by inflammatory infiltration of the capsule and peri- and para-articular tissues. In the fibrinous forms of inflammation, the fibrinous masses which often extend along the ligaments and into the joint capsule become organized, and fibrous adhesions develop between the opposed joint surfaces (anchylosis fibrosa intercartilaginea). If the articular cartilages are destroyed by severe inflammation, there develops as the inflammation subsides, first a fibrous, later a bony union of the joint surfaces (anchylosis fibrosa interossea and anchylosis ossea or synostosis).

In the treatment of arthritis one should constantly bear in mind the possibility of the development of anchylosis, and the extremity should be immobilized in the most useful position (e. g., in inflammation of the wrist and elbow joints the forearm should be immobilized in supination and slight flexion; in inflammation of the knee joint, the leg should be immobilized in the extended position; if the hip is involved the extremity should be somewhat abducted). Then the anchylosis causes the least possible disturbance of function.

Passive motion, massage, and baths are employed in the treatment of anchylosis. The object of the treatment, which is begun after the inflammation has subsided, is to obtain as free motion as possible without exciting inflammation or doing additional harm. Abnormal positions are assumed by inflamed joints early. The patient holds the limb in the position which is most comfortable and causes the least pain. Often these abnormal positions correspond to those in which, as demonstrated by Bonnet's experiments, the joint capacity is greatest.

These abnormal positions, which are called arthrogenous contractures, are caused in the beginning by reflex muscular contractions, and may be corrected under anæsthesia. Later they become permanent, and there is less and less motion as anchylosis develops. The elbow and wrist joint become pronated, the knee joint flexed, the hip joint flexed and adducted

as arthrogenous contractures and anchylosis develop. When these changes occur in the foot there is a tendency to plantar flexion.

Not infrequently pathological dislocations occur. These are due either to a distention and weakening of the capsule by the exudate (distention-dislocation), such as occur most frequently in the hip joint in typhoid, scarlet fever, and smallpox, or to the destruction of the joints and articular ends of the bones (destruction-dislocation) by severe inflammation.

FIG. 119*a*.—OSTEOMYELITIS OF THE FEMUR. Bony anchylosis. Dorsal subluxation of the tibia. (Man eighteen years of age.)

FIG. 119*b*.—SPECIMEN PREPARED AFTER AMPUTATION.

No active treatment should be employed so long as there is inflammation. Extension by weight and pulley is indicated. If there is fibrous anchylosis, the adhesions should be carefully broken up under general anæsthesia (von Langenbeck's "Brisement forcé"), and the extremity

should then be immobilized in the corrected position for two or three weeks. Contractures with bony anchylosis require a cuneiform resection of the articular ends, while in pathological dislocations a complete joint resection must be performed.

Atrophy of the muscles of the diseased extremity, particularly of the extensors, develops rapidly. The more powerful flexors then produce flexion contractures.

This atrophy is not due to the inactivity of the muscles, but, according to the Paget-Vulpian theory, is to be regarded as a reflex phenomenon (Hoffa). The irritation which affects the nerves supplying the joint is conveyed to the centers of the motor nerves in the cord, and acting upon these centers produces a simple muscle atrophy (without the reaction of degeneration).

After the inflammation has subsided, massage, active and passive motion should be employed in the treatment of the muscle atrophy.

THE HÆMATOGENOUS JOINT INFECTIONS OCCURRING IN THE COURSE OF GONORRHEA, PNEUMONIA, and TYPHOID FEVER are the most interesting.

A metastatic synovitis (arthritis) may develop at any time *during the course of a gonorrhea*, fresh or old, as soon as the diseased urethra is injured or irritated or the bacteria penetrate into the deeper layers of its mucous membrane. Pregnancy, labor, and the puerperium favor the development of this metastatic infection in woman (so-called articular rheumatism occurring in pregnancy and the puerperium). The gonorrheal conjunctivitis and stomatitis of the newborn may be accompanied by inflammation of the joints.

The gonorrheal inflammation may involve any joint. The larger joints (knee, shoulder, hip, elbow, wrist) are, however, most frequently affected. Sometimes one joint is affected, sometimes many joints simultaneously or in succession (mono- and poly-articular forms).

The knee joint is most frequently affected in men, the wrist joint in women (Nasse). Severe exertion and injuries are frequently predisposing factors.

A gonorrheal infection of a joint produces a synovitis which is accompanied by a seropurulent, serofibrinous, or serohæmorrhagic exudate. More rarely a pure serous or purulent exudate is formed. The infection may produce an inflammatory infiltration of the peri- and para-articular tissues, which is combined with a serofibrinous or fibrinopurulent exudate. The entire capsule, the ligaments, tendon sheaths, bursæ, and tissues surrounding the joint then become involved in the inflammation. The phlegmonous forms are most frequently accompanied by an inflammation of the para-articular tissues and pursue the severest course.

Pain in the joint may be the only symptom of the mildest form of

gonorrheal arthritis. The pain persists for a long time without any other symptom of inflammation, and may produce stiffness of the joint.

According to Koenig, there is apparently in these cases a fibrinous inflammation of the joint.

The inflammatory exudate in the joint and soft tissues frequently contains gonococci, as was first demonstrated by Nasse and Rindfleisch in a number of cases. It is not difficult to demonstrate gonococci if suitable culture medium is used, and recent cases, at least not older than one week, are examined.

Occasionally the ordinary pyogenic bacteria, which have entered through the diseased mucous membrane or have been introduced with an aspirating needle, are found in the exudate. They cause the phlegmonous forms of arthritis, which are often associated with general symptoms.

Gonorrheal arthritis frequently develops acutely. The suddenness with which the symptoms develop is frequently one of the best diagnostic signs. In many cases wandering joint and muscle pains are noted for some time, and then the inflammation develops subacutely in a number of joints. The gonorrheal hydrops may develop very slowly.

The fever which is almost always present in the beginning becomes high in the severest cases only and falls in a few days if the extremity is immobilized.

Gonorrheal arthritis pursues a chronic course. The hydrops is the only form which may subside rapidly, but it tends to recur. The suppurative and phlegmonous forms, on the other hand, are very resistant to treatment, although they only rarely result in abscess formation.

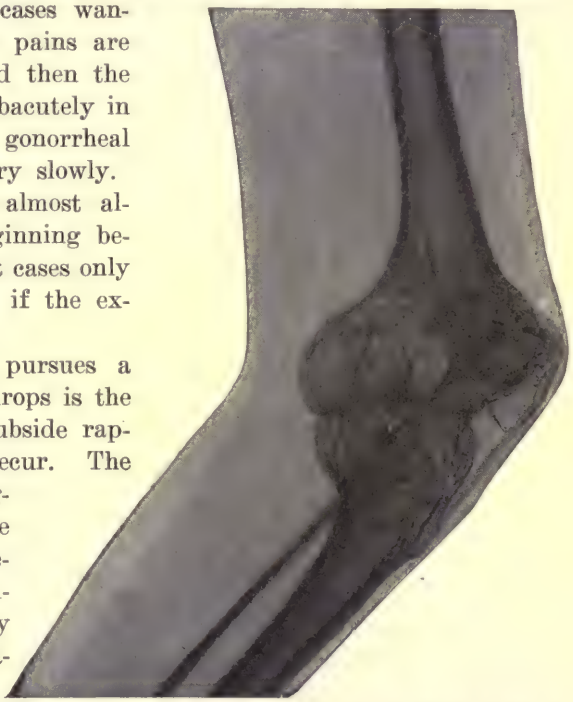

Fig. 120.—Bony Anchylosis of the Knee Joint in the Valgus Position Following a Gonorrheal Arthritis.

After one or two months the pain and swelling subside; in the meantime the muscles atrophy and the contracture of the joint, which in the beginning was mostly reflex, is followed by anchylosis.

The contraction of the infiltrated peri- and para-articular tissues, but still more the adhesions resulting from the organization of the fibrinous masses in the joint cavity, produce an anchylosis. In the beginning there may be but a partial or extensive fibrous anchylosis, but later when the articular cartilages are destroyed a bony anchylosis develops which is, as a rule, never complete, but there is no evidence upon section or in Röntgen pictures of any joint cavity (Fig. 120).

The general condition is less affected by the infection than by the severe pain.

The sequelæ of gonorrheal arthritis are, besides anchylosis, contractures and subluxations. The latter, due to a distention of the capsule and relaxation of the ligaments, may occur even after two weeks (Bennecke).

The acute onset and intense pain, which is aggravated by pressure and motion, are important in making a diagnosis of the exudative form of gonorrheal arthritis, and in differentiating it from other inflammations. The swelling which develops in the phlegmonous forms is not sharply limited, sometimes it has a doughy feel and at other times fluctuates in certain areas. The skin covering the swelling is red and œdematous. The fever, as a rule, is not high, and this often enables one to differentiate the phlegmonous form from suppurative arthritis due to the ordinary pyogenic bacteria. Often a bacteriological examination of the aspirated fluid is necessary before a differential diagnosis can be made. Fewer joints are involved in gonorrheal arthritis than in articular rheumatism, and the pain is more intense. An existing gonorrhea makes probable the diagnosis of gonorrheal arthritis. The rarer subacute and chronic forms cannot be easily differentiated from tuberculous and syphilitic arthritis.

The phlegmonous form in the chronic stage may resemble the tumor albus.

The *prognosis* as to life is good if a severe and, as a rule, fatal endocarditis does not develop. The prognosis as to function is best in the gonorrheal hydrops. The suppurative and phlegmonous forms, accompanied by an infiltration of the soft tissues, are often followed by anchylosis and joint changes.

The disease may last from four weeks to many months. The duration depends upon the severity of the infection and the number of relapses. It may recur in a joint which has already been involved, so long as the gonorrhea persists.

Absolute rest of the joint, which should be maintained as long as fever, pain, and swelling persist, is the most important part of the treatment. In most cases the inflammation will subside and contractures be prevented. Immobilizing dressings should be used for this purpose; in

inflammation of the hip these may be combined with extension. If the exudate is large, aspiration with subsequent compression may be necessary. The injection of five per cent carbolic acid (up to 8 c.c.) has been used by Koenig. Incisions should be made only when abscesses develop and in the phlegmonous forms (particularly in mixed and secondary infections).

If anchylosis develops, active and passive motion should be employed and the immobilizing dressing removed. The latter should be applied again if fever follows use of the joint. In bad cases an anæsthetic should be given when the adhesions are broken up and the contractures corrected. This procedure, which is often successful, is exceedingly painful, and cannot be satisfactorily performed unless an anæsthetic is administered.

Bier's passive hyperæmia has a favorable influence in many cases. It controls the pain and permits of early movement. The toxins are diluted by the increased transudate and are gradually absorbed.

In the lower extremity a resection of the joint may be required to correct the malposition. In the upper extremity (shoulder and elbow joints) soft tissues should be placed between the resected parts of the bone in order to obtain movement.

In rare cases *during the course of a croupous pneumonia* one or many joints may become involved. This arthritis, which develops most frequently when the disease is at its height, is caused by the pneumococcus. The serofibrinous or suppurative catarrhal synovitis (arthritis) pursues an acute course, and if there are symptoms which indicate general infection, endocarditis, or suppuration in the serous cavities, the prognosis is bad. In favorable cases the synovitis subsides after immobilization, combined with puncture and aspiration if the exudate is serous, with incision if purulent. As a rule, there is restitutio ad integrum. Pneumococcic arthritis without a preceding pneumonia is rare in adults. It is more frequent in small children, developing secondarily to foci in the articular ends of bone (*vide* Osteomyelitis). Apparently an inflamed pharyngeal mucous membrane affords the infection atrium.

Synovitis occurring during the course of typhoid fever, and caused by the typhoid bacilli, is rare. This form of arthritis develops during convalescence and pursues a benign course. The inflammation subsides after aspiration of the serous or serohæmorrhagic exudate and immobilization, if there is no mixed infection with staphylococci or streptococci which produce severe and destructive forms of suppuration.

LITERATURE.—*Bennecke.* Die gonorrhoische Gelenkentzündung. Berlin, 1899.—*Cave.* Pneumococcic arthritis. The Lancet, 1901.—*Hartmann.* Ueber die Behandlung der akuten primär synovialen Eiterungen der grossen Gelenke. Deutsche Zeitschr. f. Chir., Bd. 57, 1900, p. 231.—*Heile.* Ueber d. Zerstörung d. hyalinen Gelenkund

Epiphysenknorpels bei Tuberkulose und Eiterung. Virchows Arch., Bd. 163, 1901, p. 265.—*Hoffa.* Die Pathogenese der arthritischen Muskelatrophien. Chir.-Kongr. Verhandl., 1892, I, p. 93.—*König.* Ueber gonorrhoische Gelenkentzündungen. Deutsche med. Wochenschr., 1896, p. 751.—*Mauclaire.* Des Arthrites suppurées. Paris, 1895.—*Nasse.* Die gonorrh. Entzündungen der Gelenke u. s. w. v. Volkmanns Samml. klin. Vortr., N. F., 181, 1897.—*Pfisterer.* Ueber Pneumokokkengelenk- und Knocheneiterungen. I.-D. Berlin, 1902.—*Predtetschensky.* Akuter und chronischer Gelenkrheumatismus. Zentralbl. f. Grenzgeb., Bd. 5, 1902, p. 657.—*Schuchardt.* Die Krankheiten der Knochen und Gelenke. Stuttgart, 1899.—*Witzel.* Die Gelenk- und Knochenerkrankungen bei akuten infektiösen Erkrankungen. Bonn, 1890.

(g) PYOGENIC DISEASES OF TENDON SHEATHS AND BURSÆ

Etiology.—Inflammation of tendon sheaths and bursæ follows most frequently open injuries, penetrating foreign bodies, and the extension of inflammation from neighboring foci (suppurating wound of the skin, furuncle, subcutaneous phlegmon, erysipelas, etc.). Hæmatogenous infections are more rare. Staphylococci and streptococci are found most frequently in these inflammations; the latter especially in the severe forms. Other bacteria, such as the gonococcus, pneumococcus, bacterium coli commune, etc., are found but rarely.

Pathology.—These inflammations have an acute onset, associated with fever. In the beginning the exudate is serous, but it rapidly becomes purulent. The extension of the inflammation depends upon the anatomical relations and the size of the tendon sheaths and bursæ, and for this reason the clinical picture is often very characteristic. If the inflammation is limited to the wall of the tendon sheath or bursa, it becomes covered with granulation tissue (pyogenic membrane). If a severe inflammation produces after a few days a necrosis of the sheath or bursa, the inflammation extends rapidly and widely into the intermuscular and subcutaneous tissues. Then the clinical picture changes to that of a circumscribed or progressive phlegmon. In the former the pus gradually ruptures through the skin, and fistulæ are found which are resistant to treatment, while in the latter the inflammation extends far beyond the sheaths, the walls of which, as well as the tendons and their accessory bands, become necrotic.

The tendon is affected early by the inflammation which extends along the synovial membrane, covering the tendon and lining the sheath. The connective tissue septa become filled with leucocytes in the first few days, and karyokinetic figures and an increase of the cells in the tendon indicate a reactive growth. Necrosis of the tendon begins in from three to five days. It becomes fibrillated and necrotic. If such a tendon is not removed it acts as a foreign body, like a sequestrum in bone, and a chronic suppurating fistula develops. A little of the tendon may survive and become united with the cicatricial tissue, which develops from

the granulations after the necrotic tendon has been extruded. This cicatricial tissue interferes with the function of the part involved.

Inflammation of a bursa (bursitis acuta purulenta) is characterized clinically by a rapidly developing, painful, circumscribed, fluctuating swelling, which develops in the position of a mucous bursa (e. g., bursa præpatellaris, olecrani).

The skin covering the bursa becomes œdematous and reddened. The borders of the redness are not sharply defined. The pus either ruptures through the skin, producing a chronic fistula, or extends beneath the fascia covering the bursa and produces a large phlegmon. If the bursitis develops from a wound in the skin covering the bursa, lymphangitis and erysipelas often develop simultaneously.

Inflammation of a tendon sheath (tendovaginitis acuta purulenta) (phlegmon of the tendon sheath, panaritium tendinosum) begins with a swelling which extends rapidly along the tendon sheath and is associated with loss of function of the part involved, pain upon pressure, and motion and some reddening of the skin. Fluctuation is first elicited, when there is a large collection of pus, particularly after the process is encapsulated, and shortly before it ruptures through the skin.

The dangers and results of a suppurative bursitis lie in the extension of the inflammation to a neighboring joint. Those of a synovitis,

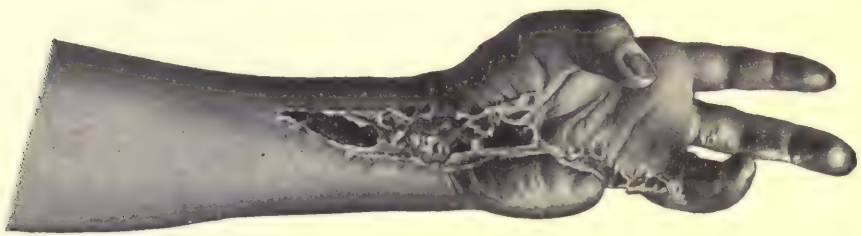

Fig. 121.—Cicatricial Contracture of the Thumb Following a Suppurative Inflammation of the Synovial Sheaths of the Flexor Tendons of the Thumb and Little Finger, the So-called V-Phlegmon. The inflammation followed a punctured wound of the little finger which was disarticulated because of osteomyelitis.

leaving out of consideration a progressive phlegmon, lie in the disturbance of function produced by the destruction of the tendons and the contractures following the contraction of the cicatricial masses. A general pyogenic infection may follow a phlegmon which develops after rupture of the synovial sheath. The diagnosis is not difficult. The position of the acute inflammatory swelling indicates with certainty that either a bursa or a tendon sheath is involved. In the latter the inflammation extends along definite anatomical routes. An inflammation of the sheaths of the flexor tendons of the second, third, and fourth fingers extends only to the transverse furrow of the palm, as the sheaths end

here. Inflammation of the sheaths of the thumb and little finger extends to the wrist joint or even higher, for frequently the sheaths of these fingers communicate with the sheath which is common to the superficial and deep flexors.

The *treatment of acute suppurative bursitis* (bursitis acuta purulenta) consists of incision and the after-treatment which is employed in suppurative inflammation. In the treatment of tendovaginitis it should be especially kept in mind that the earlier the incision is made and the pus is allowed to escape the better will be the prognosis, both as regards the repair of the tendon and the later restoration of function, as early incision prevents the formation of broad adhesions between the tendon and its sheath.

The incision should be made so that there will be the least possible cicatricial contraction. This always follows incisions made directly over the tendon which extend through the synovial sheath and its transverse connective tissue bundles. It may be avoided if small incisions are made. The incisions should be made at the side of the tendon, and the transverse fibers of the sheath and the corresponding skin (transverse furrows in the fingers, in the wrist especially the lig. carpi volare) should be avoided.

These small incisions frequently control the inflammation and permit of a complete restoration of function. When they do not provide for a free discharge of pus and control the phlegmon an incision must be made through the folds of the skin, the tendon sheaths, and the supporting ligaments of the joints. Then the fate of the tendon, associated with complete loss of function, is sealed.

If the inflammation is mild from the beginning, the extremity may be immobilized, elevated, and treated expectantly. Sometimes in staphylococcic and gonorrheal infections the inflammation subsides or an abscess forms.

Little is to be expected as regards restoration of function in the treatment of cicatricial contractures following phlegmonous tendovaginitis. The mechanical treatment, stretching of the scar, is naturally not successful, because of the anchylosis, the result of the accompanying arthritis. This treatment is rarely successful, even when the joints are not involved.

An excessive stretching of the scar may be followed by an increased contraction. The finger may be straightened by excising the scar and skin grafting the defect, but there will be no return of motion. It is rare to obtain even a little motion, for when the scar is carefully dissected away from the tendon, new adhesions develop.

Amputation is indicated if the life of the patient is threatened by general pyogenic infection or if the deformity, resulting from cicatricial contraction, interferes with work.

Acute gonorrheal bursitis and tendovaginitis should be especially mentioned. Frequently a serous, more rarely a suppurative bursitis or tendovaginitis is associated with a gonorrheal arthritis. They are, as a rule, benign; and subside spontaneously. Adhesions rarely form between the tendon and the sheath, and there is no disturbance of function.

The bursa of the tendo Achillis, the sheaths of the flexor and extensor tendons of the fingers, and the sheaths of the tendons passing behind the internal malleolus are most frequently involved (Nasse).

The extremity should be immobilized as long as the symptoms of inflammation persist. Large serous exudate should be aspirated, large purulent exudates incised, and active and passive motion should be begun at the proper time. Rapid healing and good function are obtained when proper treatment is instituted. Chronic changes with adhesions may follow this type of inflammation (Nasse).

Literature.—*v. Bergmann.* Die Behandlung der akut progred. Phlegmone. Arbeiten aus der v. Bergmannschen Klinik, Bd. 15, 1901.—*Jakobi* und *Goldmann.* Tendovaginitis suppurativa gonorrhoica. Beitr. zur klin. Chir., Bd. 12, 1894, p. 827.—*Nasse.* Die gonorrh. Entzündungen der Gelenke, Sehnenscheiden und Schleimbeutel. v. Volkmanns Samml. klin. Vortr., N. F., 181, 1897.

(h) THE PYOGENIC DISEASES OF MUSCLES AND THE SUBFASCIAL AND INTERMUSCULAR PHLEGMON

Acute Suppurative Myositis (*Myositis Acuta Purulenta*).—Acute suppurative myositis, like every pyogenic infection, may develop in different ways. The interstitial tissue, as well as the contractile substance of the muscle, may be involved. We speak of an interstitial, which is as a rule suppurative, more rarely serous or serofibrinous, and of parenchymatous-degenerative myositis.

Ectogenous infections follow most frequently wounds in which muscles have been crushed or lacerated (compound fractures, gunshot fractures with laceration and contusion of muscles, bites by animals suffering from hydrophobia, machine injuries, etc.).

Mixed infections with a number of pyogenic bacteria, also combined with putrefactive bacteria, are most frequent. If the cutaneous wound is small and its edges become quickly agglutinated, as in gunshot and stab wounds, and in fractures in which a sharp fragment pierces the skin, an inflammation of the injured muscle may not develop unless bacteria are carried into the wound by improper treatment, irrigation, and probing of the wound.

Inflammation, which is as a rule suppurative, may extend from adjacent foci. A phlegmonous erysipelas, a subcutaneous phlegmon, tendovaginitis, lymphangitis, lymphadenitis, phlebitis, osteomyelitis, etc., may

extend to the loose intermuscular connective tissues. After the fascia is destroyed the inflammation attacks the perimysium and the interstitial tissue. These are destroyed by suppuration, and the contractile substance of the muscle becomes necrotic.

A hæmatogenous infection occurring in the course of a general pyogenic infection may produce a circumscribed suppurative inflammation in one or many muscles. The muscles may be attacked simultaneously or in succession. This form of myositis is most frequently caused by staphylococci and streptococci, more rarely by the pneumococcus and gonococcus, colon and typhoid bacilli (following typhoid fever), and influenza bacilli. A subcutaneous muscle injury (laceration, contusion with hæmatoma) may suppurate if there is inflammatory focus (felon, angina) from which the bacteria, which later are deposited in the injured tissue (locus minoris resistentiæ), may be absorbed.

A *beginning myositis* is indicated by a painful swelling of the muscle, complete loss of function, and fever. The muscle involved becomes enlarged and hard, and its boundaries cannot be accurately determined because of the œdema of the surrounding structures, the subcutaneous tissue and skin. When the hard infiltrated area softens we have the symptoms of an abscess, which later ruptures through the skin.

A progressive inflammation produces, after extending to the interstitial tissue, a destruction of the entire muscle, and then extends to the surrounding tissues. Permanent loss of function and contractures follow if a fatal general infection does not develop. The severest forms of interstitial myositis are followed by large defects in the muscle which are replaced by scar tissue.

A circumscribed abscess develops if the interstitial pyogenic membrane encapsulates the pus, resulting from a destruction of the inflamed tissues. After the pus is evacuated by incision or discharged spontaneously, scar tissue fills in the defect. This scar tissue does not interfere with the function of the muscle unless the abscess has been very large.

Secondary myositis, developing from an adjacent suppurative osteomyelitis or lymphadenitis, frequently pursues a mild course with only a serous exudate, and subsides spontaneously. The formation of scar tissue and degeneration of the contractile substance (myositis fibrosa), which interfere with the function of the muscle, are the usual results.

In making a *diagnosis* it is important to note that the hard, painful swelling which develops suddenly and gradually softens, corresponds to the position of the muscle, and that the inflammation extends within the limits of the muscle. If the œdema of the surrounding tissues is marked, the development of a swelling which extends down to the bone is suggestive of myositis. A myositis may be mistaken most easily for an

inflammatory swelling associated with an inflammation of the deep lymphatics or a suppurative osteomyelitis.

The *treatment* consists of large incisions which should be made parallel to the muscle fibers. The after-treatment is carried out according to rules already given (*vide* p. 199), disturbances of function due to scar tissue may be overcome by operative measures. The distal tendon of the degenerated muscle may be cut transversely and united with the border of a healthy muscle, or with a pedunculated muscle flap taken from an adjacent muscle.

LITERATURE.—*Heinrich Lorenz.* Die Muskelerkrankungen. In Nothnagels spez. Pathologie und Therapie, Bd. 11, Wien, 1898.

Subfascial and **intermuscular phlegmons,** each of which may develop from the other, follow subcutaneous phlegmons, the rupture of suppurative inflammation of tendons, muscles, joints, bones, and infection of the connective tissue surrounding the œsophagus. These phlegmons also develop in deep wounds and in the course of metastatic infections.

They spread in the loose connective tissue, filling the intermuscular spaces, particularly along the connective tissue surrounding the large vessels (e. g., vascular sheath in the neck, axillary and popliteal fossæ).

The brawny induration of the soft tissues, the reddening and œdema of the overlying skin, fever, pain, and loss of function are the most important symptoms. In favorable cases the indurated area softens and the pus is discharged. Frequently, however, dangerous complications follow the rapid extension of the inflammation. A phlegmon of the neck may extend to the mediastinum or cause a fatal œdema of the glottis.

Large incisions should be made early. The tissues surrounding the large vessels and filling the intermuscular spaces must be exposed. If an original focus (e. g., perforation of the œsophagus, suppurative osteomyelitis or arthritis) exists, it should be found when possible. In the after-treatment, care should be exercised to prevent the erosion of large vessels. Drainage tubes should be carefully placed; hard, resistant tubes should not be used (*vide* Arteritis).

LITERATURE.—*E. v. Bergmann.* Die Behandlung der akut progredienten Phlegmone v. Bergmannsche Arbeiten, Bd. 15, 1901. Berlin, Hirschwald.

Woody phlegmon (brawny induration) of Reclus is a peculiar inflammation, which involves most frequently the intermuscular and subcutaneous tissues of the neck. It pursues a chronic course, with little fever, a boardlike hardness, and almost painless swelling of the soft tissues.

There is but little tendency to suppuration.

Streptococci, staphylococci, pneumococci, and diphtheria bacilli have been found in the exudate, which is small in amount. Sometimes bac-

teria are looked for in vain. These inflammations develop most often from the mucous membrane of the floor of the mouth and pharynx. Apparently they are caused by attenuated forms of bacteria, and for this reason the tissues do not become necrotic and pus is not formed, or only in small amounts.

If an incision is made early, because of the danger of œdema of the glottis or dysphagia, the surgeon finds a brawny indurated connective tissue, and often in the intermuscular spaces a cloudy, many times a purulent exudate, especially in the submaxillary region adjacent to swollen and softened lymph nodes. This form of inflammation may be mistaken for actinomycosis, as the boardlike infiltration is the most important characteristic of the latter.

Warm, moist compresses are to be recommended to soften the infiltrated area. Quicker results are obtained by incision and exposure of the infiltrated intermuscular spaces. Even if pus is not found the bacteria are removed with the wound secretion.

LITERATURE.—*Jansson.* Holzphlegmone. Hygieia, January 2, 1904.—*Kusnetzoff.* Ueber die Holzphlegmonen des Halses (Reclus). Arch. f. klin. Chir., Bd. 58, 1899, p. 455.

(i) PYOGENIC INFECTIONS OF SEROUS CAVITIES AND DIFFERENT ORGANS

Infection of the serous cavities and viscera may occur from without (ectogenous) or from within (endogenous). Ectogenous infections follow penetrating or perforating wounds (deep cuts, stab and gunshot wounds); endogenous infections follow traumatic or inflammatory perforation of organs lined with mucous membrane (e. g., peritonitis following subcutaneous rupture of the intestine, pleuritis following traumatic rupture of a bronchiole in subcutaneous fracture of the ribs, meningitis after injury of the internal ear, or ethmoid cells in fracture of the base of the skull, etc.).

Such an infection may occur through the lymphatics (lymphogenous) or by direct extension (e. g., pleurisy secondary to lung abscess, pneumonia, osteomyelitis of a rib, peritonitis; meningitis secondary to osteomyelitis of the cranial bones, thrombophlebitis of the sinuses, brain abscess; peritonitis secondary to pleuritis, the inflammation extending along the lymphatics of the diaphragm, phlegmon of the stomach, intestines, and abdominal wall; brain abscess secondary to extradural suppuration or thrombophlebitis of the veins of the diploë; abscess of the kidney (pyonephrosis) secondary to cystitis).

Hæmatogenous infection may involve the viscera and serous cavities. In general pyogenic infections, accompanied by metastatic inflammations, they become infected at the same time that other tissues do. In-

fected emboli lodge in the lung and cause lung abscess; infection of the liver occurs through the portal vein.

Three principal forms of inflammation, which may be accompanied by different exudates, are to be differentiated in serous cavities: the *circumscribed*, the *acute progressive*, and *general inflammation*. In the circumscribed form the serous surfaces become adherent at the borders of the granulation tissue. In the acute progressive form the inflammation is not encapsulated, or only incompletely. An encapsulated focus may also rupture through the protecting granulation tissue; then an acute progressive inflammation develops. The general form in which the entire surface of serous membranes is involved develops from the acute progressive forms.

Following pyogenic infections of the viscera, circumscribed foci, which are usually multiple, may develop, or the inflammation may be diffuse involving the entire viscus.

The clinical course, diagnosis and treatment of these infections belong to the province of special surgery.

When suppuration occurs in serous membranes a wide incision should be made, and free drainage established as soon as possible. In the skull and thorax the necessary preliminary operation must be performed (trephining, resection of rib). In the milder forms of inflammation puncture with aspiration may be sufficient (e. g., in pleurisy, lumbar puncture in meningitis). When abscesses develop in the different viscera they should be incised; when possible, and the conditions found indicate such a procedure, the entire viscus should be extirpated (e. g., kidney, testicle, ovary, spleen).

LITERATURE.—*Haegler.* Ueber das freie seröse Exsudat des Peritoneum als Frühsymptom einer Perforationsperitonitis. Zentralbl. f. Chir., 1904, p. 282.—*Noetzel.* Die Prinzipien der Peritonitisbehandlung. Beiträge z. klin. Chir., Bd. 46, 1905, p. 514; —Die Behandlung der appendizitischen Abszesse. Ibid., Bd. 47, 1905, p. 826.—*Peiser.* Zur Pathologie der bakteriellen Peritonitis. Ibid., Bd. 45, 1905, p. 111.

CHAPTER V

GENERAL PYOGENIC INFECTIONS

DURING the course of any local pyogenic infection micro-organisms and their toxins may be absorbed, and groups of bacteria may invade the lymphatic vessels and blood vessels and gain access to the blood. A general reaction follows the absorption of infectious materials, which

varies in intensity, depending upon the number and virulence of the bacteria absorbed and the character of their toxins. This general reaction is characterized by fever and the symptoms which accompany it.

The general reaction following a local infection is due to the absorption of toxins, and is apparently of a protective nature. Bacteria are found in the blood quite frequently even in the mild general reactions, but the latter are very different from general pyogenic infections. In the general reaction following a local infection the bactericidal properties of the blood and tissue fluids destroy the bacteria and neutralize the toxins, while in the general pyogenic infections the resistance of the organism is so reduced, or the bacteria are absorbed in such large numbers and are so virulent, that they multiply rapidly and may be deposited in the different tissues and viscera producing metastatic foci. If the bacteria and their toxins prevail over the bactericidal properties of the blood and tissue fluids, a general pyogenic infection develops.

There are two principal forms of general pyogenic infections, between which there are many transitions. The chief characteristic of one form is the development of multiple suppurating metastatic foci, of the other the multiplication of bacteria in the blood without the development of metastatic foci. I therefore differentiate:

A *general pyogenic infection* with *metastases* in which there occur *intermittent transitory infections* of the *blood* (metastatic infection) and

A *general pyogenic infection* without *metastases* in which there is a *persistent* (*toxic* and *bacterial*) *infection* of the *blood.*

In the general infection characterized by metastases the infection is spread by way of the blood stream only, and foci of infection develop in different parts of the body. The infection may be produced in two ways: Either *groups* of *bacteria* (bacterial emboli) pass through the walls of the *diseased vessels* in the primary focus into the blood stream, or are carried by *pieces* of *thrombi* (infected emboli) which have been destroyed by suppuration into the circulation, producing where they are deposited metastatic foci of suppuration. Infarction frequently precedes the development of these foci.

In some cases the bacteria and their toxins are not found constantly in the blood, occurring only when the bactericidal substances of the latter have been exhausted. This may happen daily or after long intervals, recurring regularly or irregularly. When the bactericidal substances are formed again the bacteria are removed from the blood and deposited in large numbers in different parts of the body (bone marrow in children, joints, and large viscera) where they incite processes which combat the infection.

Etiologically every *hæmatogenous infection* (hæmatogenous osteomyelitis, arthritis, etc.) must be regarded as a mild form of *general pyogenic*

infection with *metastases*, regardless of whether or not a primary infection atrium can be found. Clinically a single metastasis occurring in the cases above cited can only be regarded as part of a general infection, when the blood infection persists and there are other symptoms indicative of a general infection.

The form without metastases is a persistent general infection with bacteria and their toxins. Theoretically a general *bacterial infection* (bacteriæmia) may be differentiated from a general *toxic infection* (toxæmia), depending upon whether the bacteria or their toxins predominate in the blood. Such a distinction cannot, however, be made clinically.

In *general bacterial infections* the bacteria which are absorbed from the primary focus rapidly multiply in the blood, for the organism is not able to produce enough protective substances to destroy the bacteria and neutralize the toxins (septicæmia as defined by bacteriologists). The resistance of the organism is so low relative to the virulence of the bacteria that there is no inflammatory reaction in the tissues ending in pus formation.

In the *general toxic infections* (such as occur in tetanus and diphtheria) large quantities of toxins enter the circulation. This form of infection, which can scarcely be distinguished from the bacterial infections except by negative blood cultures, occurs more frequently in infections with the uncommon pyogenic bacteria and in mixed infections, especially in mixed infections with putrefactive bacteria.

There are a number of transitions between these two principal forms of general pyogenic infections.

It is not necessary that each of these transitional forms be named, and besides, it is frequently difficult to apply a term which accurately describes the condition. The terms which have been introduced into the nomenclature of the different forms of general infections have been variously interpreted by different authorities, and this has resulted in considerable confusion; for example, Gussenbauer and Brunner regard sepsis as synonymous with putrefaction, while Canon and Lenhartz regard it as synonymous with general pyogenic infection. Von Kahlden regards septicæmia as synonymous with toxæmia (regardless of whether it is due to pyogenic or putrefactive bacteria). In order to avoid confusion and simplify matters, Lexer employs the term putrid infection and general putrid infection for local inflammatory process characterized by putrefactive changes and the general symptoms following them.

While the terms pyæmia, septicæmia, sapræmia, etc., are not used in the German edition of this book, it seems best to explain the significance of these terms, which have become so firmly established in American medical literature. The *general pyogenic infection with metastases* as

used by Lexer is synonymous with *pyæmia*; *general pyogenic infection without metastases* with *septicæmia*, and *general putrid infection* with *sapræmia*.

(a) GENERAL PYOGENIC INFECTIONS WITH METASTASES (PYÆMIA)

Bacteria Most Commonly Found.—Any variety of pyogenic bacteria from any inflammatory focus is able to produce metastatic suppuration in the body. Staphylococci are found most frequently in these general infections, and next in order of frequency, streptococci, which tend to produce suppuration of the joints and serous membranes, phlegmons, and erysipelas. Pneumococci, gonococci, colon and typhoid bacilli (after typhoid fever) are found less frequently than staphylococci and streptococci. Mixed infections with the staphylococci and streptococci are relatively common.

Most Common Sources of Infection.—The bacteria may be absorbed from any inflammatory focus or may be carried from diseased vessels in the form of bacterial or infected emboli into the general circulation. The most common sources of infection are: 1. Infected wounds. 2. Local inflammatory processes. 3. Infected ulcers. Operation-wounds at the present time are rarely the source of general infection, although in pre-antiseptic times severe general infections were exceedingly common secondary to infected operation-wounds.

Infected wounds with recesses and pockets, the tissues of which are infiltrated with blood and necrotic (e. g., contused and lacerated wounds, large wounds of the soft tissues surrounding complicated compound fractures, bites inflicted by mad animals, deep wounds following explosions and machine injuries, and the inner surface of the uterus after delivery), are most frequently followed by general infections. Putrefactive bacteria, which are often associated with pyogenic bacteria in infections of this character, increase the virulence of the latter and favor the development of general infections.

Any local inflammatory process such as a furuncle, a phlegmon, an inflammation of a mucous membrane (especially an angina, an empyema of one of the accessory sinuses of the nose, otitis media), lymphangitis, thrombophlebitis, hæmatogenous endocarditis, arthritis, and suppurative osteomyelitis, may be the source of a general pyogenic infection.

Ulcers of all sorts (tuberculous, typhoid, syphilitic, carcinomatous) may provide the infection atrium for a general infection.

The So-called Cryptogenic Infections.—Although there are cases in which the primary focus cannot be found, it is certain that one exists or has existed in each case. In the cases of so-called cryptogenic infec-

tion there may have been a slight inflammation of a mucous membrane which gave rise to but few symptoms, or a furuncle which has healed, and although the lesion may have healed entirely, bacteria may have been retained in the thrombosed veins of the area involved or the lymphatic nodes which drained it and later have caused the general infection.

Factors Favoring the Development of a General Infection.—All those factors which favor the absorption of bacteria and their toxins, or the setting loose of particles of thrombi or groups of bacteria in the lymphatic vessels and veins favor the development of a general infection. The most common are injuries and mechanical irritation of the wounds, which even at the present time are frequently not avoided in the treatment.

Trauma and other injuries, providing as they do the locus minoris resistentiæ, also determine the localization of hæmatogenous infections. Lacerations, hæmatomas, and circulatory disturbances so reduce the resistance of the tissues that they are no longer able to destroy, as the normal tissues do, the bacteria which are deposited by the blood stream. To cite an example of the lower resistance of tissues following an injury, a subcutaneous fracture, a subcutaneous or muscular hæmatoma becomes infected during the course of an angina and suppurates.

Susceptibility of Different Tissues and Viscera.—The different tissues and viscera of the body must have some peculiar properties, concerning which but little is known, which determine the localization of hæmatogenous infections. For example, if a small culture of pyogenic bacteria is injected into the cutaneous vein of an experimental animal, suppurating foci develop first in the lungs, in which the grosser particles which have been injected are retained. Later foci develop in the joints or in young animals in the bone marrow, then in the periosteum and kidneys, less frequently in the liver and serous cavities. Only after the injection of large amounts of a culture do foci develop in other organs, especially in the muscles, and finally in the myocardium, subcutaneous tissues, etc.

Mechanical conditions seem to play an important part in determining the localization of infections, for the bacteria, especially if carried in infected emboli, lodge most frequently in very vascular organs (lungs, bone marrow, synovial membranes, kidney, and liver). Bacteria seem also to be deposited in tissues and viscera (bone marrow and spleen) which form large amounts of bactericidal substances. They are retained, and if too many and too virulent bacteria are not deposited they are destroyed.

Symptoms.—Clinically the symptoms of the general reaction (excepting, of course, the so-called cryptogenic infections) which accompany an infected wound or a local inflammatory process precede the symptoms

of the general infection. The fever accompanying the general reaction, which often begins with a chill and rises abruptly or gradually, is called *absorption fever* to differentiate it from the fever occurring in the general infections into which it often passes imperceptibly. This absorption fever should not be regarded, however, as indicative of a general infection unless there are other symptoms which usually occur in these cases.

General infection with metastases is characterized by frequent chills, intermittent fever, severe general symptoms, and the development of metastases.

The disease begins with a severe chill, and during the subsequent course of the infection chills may recur many times during the day or after long intervals. A high temperature (103°–104° F.) which rises abruptly accompanies each chill. The temperature drops suddenly from three to four degrees each day, especially in the morning, and rises later in the day, but this rise is not accompanied by a chill. The fever accompanying these infections is therefore usually of either an intermittent or remittent type. There are, however, frequent exceptions.

Character of the Fever.—The fever becomes continuous if there is a rapid and continuous absorption of pyogenic substances from an extensive primary or secondary suppurating focus, or if the protective substances are so reduced that the blood infection is frequently repeated or becomes permanent. These bactericidal substances, when formed in sufficient amounts, destroy the bacteria and neutralize the toxins so that the blood infection is, as a rule, transitory (*vide* Fig. 124).

A remittent fever should not be regarded as indicative of a metastatic infection unless there are other symptoms. A simple absorption fever which is designated by many authors as "septic fever" or even "sepsis" may have a similar curve, as is frequently the case in inflammations caused by streptococci (*vide* Fig. 126).

One should not conclude, as, for example, in erysipelas, that there is a toxic or bacterial blood infection, because there is a continuous absorption fever, even if bacteria may be cultivated from the blood. So long as the other symptoms are wanting the fever is merely indicative of a long persisting reaction of the organism to the substances which are absorbed from the inflammatory focus.

Different types of fever are produced by different bacteria. These differences are so slight and depend upon so many factors that they have no clinical value.

The general symptoms are malaise, pain in the extremities, rapid pulse and respiration, dry tongue, thirst, dry hot skin or profuse perspiration, headache, stupor, delirium, anorexia, and vomiting. These symptoms are common to the infectious diseases.

Associated with these are symptoms which also occur in general infections without metastases: severe diarrhœa, which is caused by the excretion of the absorbed toxins or metastatic inflammation of the intestinal mucous membrane due to emboli; icterus, which may develop after a few days, probably due to the destruction of red corpuscles (hæmocytolysis); acute splenic swelling which develops in this form of infection and in other infectious diseases, sometimes produced by metastatic abscesses; alterations in the composition of the blood shown by a reduction in the number of red blood corpuscles, and frequently by quite a marked leucocytosis (*vide* Blood Infection); and finally ulcerative endocarditis, which, according to Lenhartz, develops in from one fourth to one fifth of all cases of general infection, especially in the form accompanied by metastases. The ulcerative endocarditis develops most frequently upon the left side of the heart.

If, as is frequently the case, the symptoms of lung involvement due to metastatic abscesses and inflammatory infiltration are most prominent, the patient becomes dyspnœic, coughs and expectorates considerable foul-smelling material, which may differ quite a great deal in character. Frequently during the later course of the infection the physical findings of a pleurisy developing from a focus situated in the periphery of the lung may be elicited. The development of an infarct of the lung is indicated by sudden dyspnœa, cardiac weakness, and bloody sputum.

Involvement of the kidneys may not be accompanied by symptoms or may be indicated by the symptoms of a severe acute nephritis. Small foci resembling an infarct in shape develop in the cortex. Striated foci also develop in the medulla. The latter are caused by the bacteria which are filtered through the glomeruli into the uriniferous tubules.

One after another of the joints, especially the larger ones, may become affected. Sometimes the synovitis develops acutely, sometimes subacutely. The exudate may be serous, serofibrinous, serohæmorrhagic, or purulent. In severe cases the inflammation extends rapidly to the capsule and the articular cartilages, which are destroyed. Besides these joint metastases, there is often found (especially in general infections due to gonococci) inflammation of the tendon sheaths and bursæ.

Frequently small and large hæmorrhages occur in the eye, the retina becomes necrotic; the crystalline lens becomes clouded or suppurates, iridocyclitis and panophthalmitis develop.

The serous membranes, pia mater, pleura, pericardium, peritoneum, become infected through the blood or by extension of some focus in the brain, lung, myocardium, or abdominal viscera. In rare cases the tunica vaginalis testis becomes inflamed. As a rule, the inflammation is secondary to some focus in the testicle or epididymis.

Muscle foci sometimes produce circumscribed abscesses, at other times

progressive phlegmons. These foci are especially numerous in staphylococcic infections, and often precede the development of subcutaneous abscesses. In chronic cases frequently sixty to one hundred subcutaneous and muscular abscesses develop and must be opened. The subcutaneous tissue may be the seat of progressive phlegmons, particularly if the infection is due to streptococci, while metastatic erysipelas, as well as a scarlet-fever-like erythema, small hæmorrhages, herpes, and pustules may develop in the skin.

The metastases stand in different relations to the primary focus. The metastases are spoken of as secondary, tertiary, etc., depending upon whether they have developed from the primary or a metastatic focus. An ulcerative endocarditis is very frequently the origin of such metastases.

A general infection with metastases may pursue an acute or chronic course. The acute forms prove fatal in a short time if the formation of metastases is not prevented by proper treatment of the wound which is in the infection atrium. The greater the number of metastatic foci, the greater are the dangers of a permanent blood infection. Bacteria and their toxins pass into the blood from all these foci.

Chronic Forms of Infection.—The chronic forms which are sometimes observed in staphylococcic infections may continue for weeks and months. In rare cases, if there is no severe infection of the viscera, recovery may occur even after a number of muscular and subcutaneous abscesses and suppurative arthritis have developed.

Prognosis.—The prognosis is gravest in the acute cases with multiple metastases and pathological changes in the important viscera. Healing may occur in the chronic cases with a limited number of metastases.

Diagnosis.—The diagnosis in advanced cases is not difficult, especially if the original inflammatory focus is still present. In the cryptogenic cases the pathological changes in the viscera often are most prominent and the disease may be mistaken for suppurative nephritis, cerebrospinal meningitis, or endocarditis occurring in the course of acute articular rheumatism. If the symptoms are not pronounced, acute miliary tuberculosis and typhoid fever must be considered.

Blood Examination.—Examination of the metastatic foci and of the blood is most important in making a diagnosis. Bacteria may be demonstrated in the blood in a large number of cases shortly after the chill with the methods which are employed at the present time. The finding of pyogenic bacteria excludes acute articular rheumatism, for in rheumatism proper bacteria are never found (Lenhartz).

Treatment.—The first indication in the treatment of general infection with metastases is to prevent further infection of the blood. Often this is accomplished by thorough exposure of the primary focus accord-

ing to the general rules already given (*vide* p. 198). Often amputation of the diseased extremity or extirpation of the diseased viscus (e. g., kidney) is required. In other cases the extension of the infection by the separation of thrombi in an inflamed vein is prevented by the ligation of the large trunks, e. g., ligation of the internal jugular vein in carbuncle of the face with thrombophlebitis of the anterior facial vein or in thrombosis of the transverse sinus secondary to otitis media; ligation of the long saphenous vein in thrombophlebitis of its branches (cf. Suppurative Phlebitis).

The second indication is to expose all demonstrable and accessible metastatic foci and to treat them according to the general rules.

The third indication is to improve the general condition of the patient by a nutritious but light diet and to stimulate the heart (particularly by subcutaneous injections of camphor oil) when it begins to fail (cf. General Treatment of Blood Infections, p. 291).

(b) GENERAL PYOGENIC INFECTION WITHOUT METASTASES (SEPTICÆMIA)

In the general pyogenic infection without metastases, the blood and with it the entire organism is flooded with bacteria and their toxins. The bacteria multiply rapidly in the blood and the tissues. A bacterial infection develops when the bactericidal substances are no longer produced, or in small amounts only, or when the invading bacteria are especially virulent.

Factors Favoring Development of General Infection.—The factors which favor and cause the development of this form of infection are the same as described in the general infection with metastases with the following exceptions: 1. In the pure type the infection is not carried by emboli. 2. The development of a primary inflammatory focus before the beginning of the general infection is not essential. The local and general symptoms may develop simultaneously after the infection has been introduced (e. g., after injury received while performing a post-mortem examination).

The streptococcus pyogenes is found most frequently in this type of infection. The streptococcus may enter the blood not only from pyogenic, but from putrefactive wound infections as well. Its virulence is increased by symbiosis with putrefactive bacteria. Staphylococci, pneumococci, colon bacilli, and other bacteria are more rarely the cause of general infections. Pneumococci are found most frequently in general infections following pneumonia. In mixed infections with the streptococcus and some other variety of pyogenic bacteria, the former alone, as a rule, passes into the blood.

Relative Frequency with which Different Bacteria Cause Metastatic Suppuration.—A good idea may be had from Lenhartz's statistics of the relative frequency with which general infections produced by the different bacteria pursue their course with and without metastases. For sake of convenience the cases are arranged in the following way:

Bacteria.	No. of cases.	Without Metastases.	With Metastases.
Streptococci	100	65%	35%
Staphylococci	22	5%	95%
Pneumococci	20	75%	25%
Bacterium coli commune	8	78%	22%

The general pyogenic infection is, as a rule, a bacterial one, as the blood contains large numbers of rapidly multiplying bacteria which produce toxins. If bacteria cannot be demonstrated in the blood, and if the primary infection is purely pyogenic, one may conclude that the symptoms are produced by toxins which are absorbed from the primary focus. This form, known as the general toxic infection, is commonly found in putrefactive infections, but is the exception in pyogenic infections.

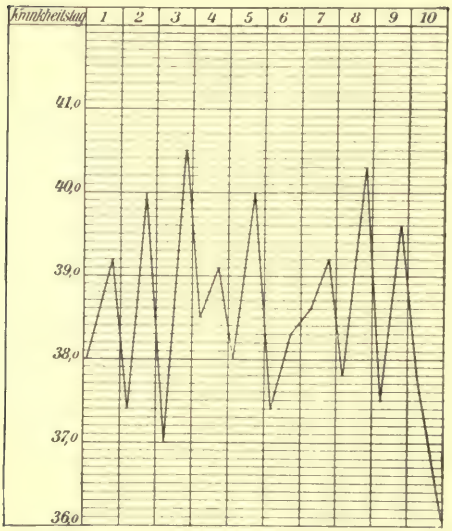

Fig. 122.—Infection Forming Metastases (Staphylococcus Aureus Found in Local and Metastatic Foci) Following Suppurative Thrombophlebitis Involving the Long Saphenous and Femoral Veins Secondary to a Varicose Ulcer of the Leg. Death on tenth day following admission to the hospital. Post-mortem examination, abscesses of the lungs, pleurisy, abscesses in the muscles, foci in the kidneys, metastases in two of the joints.

Symptoms.—The symptoms may develop within a few hours, often after an apparently insignificant injury. The disease begins with a chill, high fever, and severe general symptoms (especially after injuries received in post-mortem examinations of patients dying of acute suppurative or general infection). Sometimes the symptoms develop gradually after an absorption fever, resulting from a local suppurative inflammation (e. g., phlegmon, peritonitis, arthritis, osteomyelitis), has persisted for some time. Then there is nothing to

indicate when the general infection begins, for, as a rule, there is no chill in these cases. All the symptoms which occur in infectious diseases may be found in this type of infection. The temperature continues high (104° F. and higher), with but little variation, ½° to 1°. Before death the patient may pass into collapse.

In the beginning the symptoms are depression, weakness, restlessness, cramplike pains in the extremities, anorexia, nausea, and vomiting. The pulse and respiration then become more rapid, and cerebral symptoms (headache, delirium, coma, stupor) develop. A hot dry skin, dry tongue, cracked lips, great thirst, small amounts of concentrated urine rich in albumin, a persistent, at times, bloody diarrhœa, cyanotic appearance, icterus, pustular eruption, or one resembling scarlet fever or urticaria, small hæmorrhages into the skin (petechiæ), which are caused by diapedesis of blood through the walls of the capillaries altered by toxins, splenic swelling, involuntary discharge of fæces and urine, tendency to formation of bedsores over sacrum, trochanter, and heel complete the clinical picture. The heart gradually fails and death occurs.

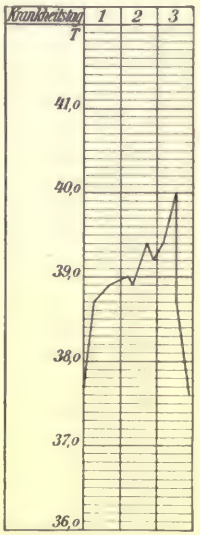

FIG. 123.—GENERAL PYOGENIC INFECTION WITHOUT METASTASES (STREPTOCOCCI IN THE PUS AND CIRCULATING BLOOD) FOLLOWING A SEROPURULENT PHLEGMON OF THE SHEATH OF THE LONG FLEXOR TENDON OF THE THUMB, SECONDARY TO A PUNCTURED WOUND OF THE THUMB. Multiple and extensive incisions. Death on the third day with abrupt fall of temperature.

Appearance of the Wound.—The appearance of the wound is characteristic. The wound surfaces, which are dry and discolored, are covered with a membrane, resulting from a superficial necrosis of the tissues and a fibrinous exudate which contains bacteria. Ragged accidental wounds and smooth incisions have this same appearance. The wound surfaces are no longer able to secrete pus or form granulation tissue. If such a wound is adjacent to a subcutaneous fracture, frequently a suppurative inflammation develops in the latter.

Changes in the Blood.—As a rule, there is a marked reduction in the number of red corpuscles (Grawitz observed in one case a reduction of ninety per cent).

Large amounts of albumin are lost and the blood becomes hydræmic.

As a rule, there is no leucocytosis. The vessel walls are altered in the severe cases, and spontaneous hæmorrhages into the skin, serous membranes, conjunctiva, retina, and bone marrow occur (hæmorrhagic general infection).

Transitional Forms.—In the transitional forms, which are not rare, there are a number of other symptoms. These symptoms, which generally begin with a chill, vary, depending upon the position of the metastatic inflammation. They are caused in part by an endocarditis.

Prognosis.—As a rule, the disease runs a rapid course. The severest cases terminate fatally in one or two days (particularly puerperal infections) (Fig. 123). Frequently there is some improvement after operation, such as incision of the phlegmon, amputation of the infected extremity, etc. After a few days, however, the disease may progress with renewed vigor, indicating how futile operative interference has been. When the disease is well advanced, it is seldom possible to save the patient.

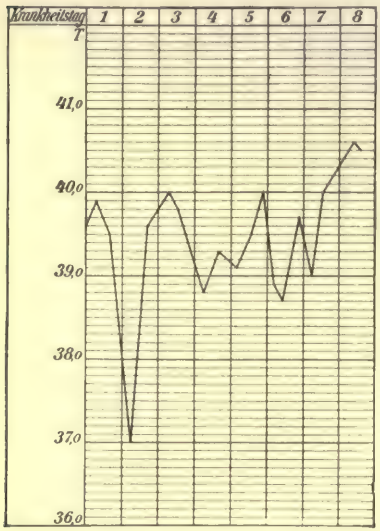

FIG. 124.—INFECTION FORMING METASTASES WITH TRANSITION INTO A GENERAL BLOOD INFECTION. CAUSED BY STREPTOCOCCI FOLLOWING AN INFECTION OF THE SYNOVIAL SHEATH OF THE FLEXOR TENDONS OF THE LITTLE FINGER AND FOREARM SECONDARY TO A CRUSHING INJURY. First day: fall of fever after incision. Second day: chill followed by pain in the knee joint (seropurulent synovitis). Fifth day: streptococci numerous in the blood, in the exudate removed from the joint, and in the pus discharged from the phlegmon. Seventh day: streptococci very numerous in the blood. Eighth day: death.

Diagnosis.—The diagnosis is not difficult when the symptoms are pronounced. Repeated blood examinations prevent mistaking this disease for typhoid fever and miliary tuberculosis. It is most difficult to differentiate between scarlet fever with secondary streptococcic infection and a general streptococcic infection with a scarlatiniform exanthem.

The diagnosis is based in the beginning upon the severity of all the symptoms, and not upon a positive blood finding alone. It is frequently difficult to make a diagnosis between a general infection and sapræmia. If the daily blood examinations are positive, and bacteria are found in large numbers, the diagnosis of general infection may be made. The characteristic appearance of the wound and the frequent absence of leucocytosis indicate that the resistance of the patient is greatly reduced.

There is little clinical difference between a general pyogenic and a putrefactive infection. The symptoms and the clinical course are the same. The chief distinction is this: that the local focus presents in one suppurative, in the other putrefactive changes. It frequently happens,

however, that a general streptococcic infection develops from a putrefactive focus.

The prophylactic treatment has already been discussed in the chapter dealing with the treatment of pyogenic infections (*vide* p. 201).

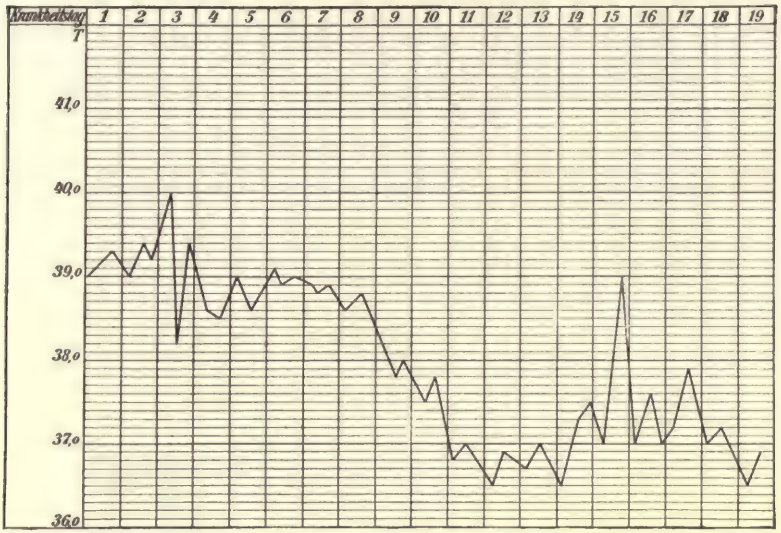

FIG. 125.—INFECTION FORMING METASTASES FOLLOWING CARBUNCLE OF THE UPPER LIP; ATYPICAL FEVER FROM FOURTH TO TENTH DAY. First day: incision. Second and third days: chills. Third day: large incision, opening of the anterior facial vein which contained a suppurating thrombus and ligation of the internal jugular vein. Fourth to tenth day: absorption fever, gradually falling as the conditions in the wound improve and the inflammation subsides. Fifteenth day: chill and development of small abscess in an infected muscle. Recovery.

The chief indication is to expose by early incision or to remove by amputation or joint resection the original focus. This is only possible if the case is seen early. The expectant treatment should not be employed (*vide* Phlegmon, p. 212).

The remaining treatment is palliative. Narcotics are given for the cerebral symptoms. Comatose and delirious patients should be fed artificially. The strength of the heart must be sustained. Subcutaneous injections of physiological salt solution (0.9 per cent) act more rapidly and favorably than any other agent. In adults 1 to 2 l., in children 200 c.c. may be given one or many times daily. It improves the general condition, increases the excretion of urine, and in this way favors the excretion of the toxins. Serum treatment has met with as little success as the injection of bactericidal substances, such as sublimate (Bacelli and Kazmarsky) and silver preparations (Credé), into the blood. Brunner and Cohn have demonstrated that the latter have

no action when employed in the treatment of experimental general infections.

LITERATURE.—*B. Blohm.*—Ueber Vereiterung subkutaner Frakturen. I.-D. Berlin, 1898.—*K. Brunner.* Wundinfektion und Wundbehandlung. Part III: Die Begriffe Pyämie und Sephthämie im Lichte der bakteriologischen Forschungsergebnisse. Frauenfeld, 1899;—Ueber das lösliche Silber und seinen therapeutischen Wert. Fortschritte d. Mediz., 1900, No. 20.—*Busse.* Ueber Sacharomycosis hom. Virchows Archiv, Bd. 140, 1895, p. 23.—*E. Cohn.* Ueber den antiseptischen Wert des Argentum colloidale Credé und seine Wirkung bei Infektionen. I.-D. Königsberg, 1902.—*Gussenbauer.* Sephthämie, Pyohämie und Pyosephthämie. Deutsche Chir., Stuttgart, 1882. —*Hentschel.* Beitrag zur Lehre von der Pyämie und Sepsis. Festschr. f. Benno Schmidt, Leipzig, 1896, p. 121.—*v. Kahlden.* Ueber Septikämie und Pyämie. Zentralbl. f. Pathol., 1902, p. 783.—*Muscatello* und *Ottaviano.* Ueber die Staphylokokken pyämie. Virchows Arch., Bd. 166, 1901, p. 212 and Lit., p. 255.—Further Lit., p. 261.

Blood Cultures.—Blood examinations made during life should determine the variety of bacteria in the blood and give some idea as to their number.

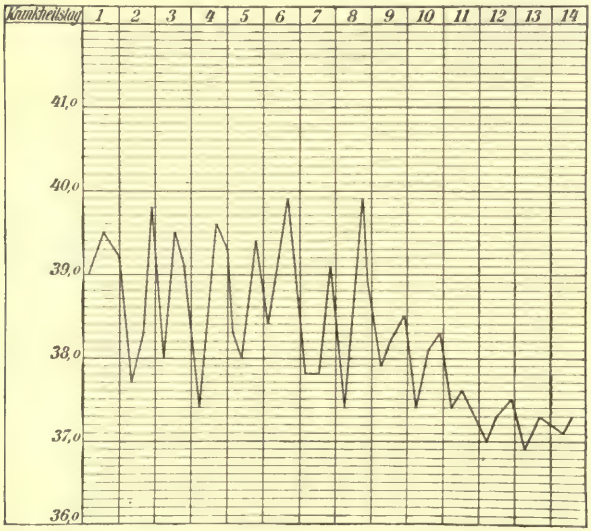

FIG. 126.—ABSORPTION FEVER (WITH SEVERE SYMPTOMS, WITHOUT CHILLS) OCCURRING IN A PURULENT ARTHRITIS OF THE KNEE WHICH FOLLOWED A STREPTOCOCCIC OSTEOMYELITIS INVOLVING THE ARTICULAR END OF THE FEMUR. Fourth day: incision of the joint. Fifth day: laying open of the joint. Sixth day: a few streptococci in the blood. Seventh day: resection of the knee joint (metaphyseal focus posteriorly which had ruptured into joint). Ninth day: fall of temperature and recovery after some weeks without a recurrence of fever.

Aseptic preparation of the field surrounding the vein from which the blood is to be taken must be as nearly perfect as possible. In Sittmann's method the skin covering the median basilic vein is thoroughly sterilized and a slight venous stasis is produced by applying an elastic constrictor loosely about the upper arm near the axilla. In children a branch of the long saphenous vein may be chosen. A needle, attached to a Pravaz syringe—or better, a larger syringe which holds 10 c.c.—is passed obliquely through the skin into the vein. The needle should be passed in the direction of the blood stream. As

a rule, the piston of the syringe is forced outward by the pressure of the blood streaming into the needle.

About 5 c.c. are taken from small children, 10 c.c. or more from adults. The blood is carefully protected from contamination and is used in the following ways:

One c.c. should be injected into the subcutaneous tissues or peritoneum of white rats. Death follows in one or two days if the blood contains streptococci or pneumococci. If it does not occur, it is not proof positive that there are no bacteria in the blood.

One c.c. is poured into a flask containing 10 c.c. of gelatin, and the gelatin then liquefied by warming the flask over a water bath. After the blood and gelatin are thoroughly mixed, the mixture is poured into a sterile Petri dish. If kept at 22° C. in an incubator or at room temperature colonies develop in from twelve to twenty-four hours if bacteria are present. The number of colonies in 1 qcm. gives a good idea of the total number of bacteria. The variety of bacteria may be determined by inoculations or by microscopic examination.

One c.c. of blood may be mixed with 10 c.c. of nutrient bouillon. If kept at 37° C. in the incubator even a few bacteria multiply so rapidly in twelve hours that they may be transferred to agar or gelatin with a platinum loop. A luxurious growth can be obtained in this way. Anærobic cultures media must be used when the primary infection is caused by putrefactive bacteria.

Blood examinations made after death are of little value. Bacteria pass from the suppurating focus or from the intestines into the blood during the death agony. In cases in which the cultures made during life were negative, the conclusion may be drawn, if large numbers of bacteria are found in the blood immediately after death, that a few were present during life.

CHAPTER VI

PUTREFACTIVE INFECTIONS

(a) PUTREFACTIVE WOUND INFECTIONS AND GENERAL INFECTIONS

As suppurative inflammation is the chief characteristic of the activity of pyogenic bacteria, so putrefactive inflammation with the formation of an exudate, which in the beginning is serohæmorrhagic in character and later becomes discolored and foul smelling, contains gas and is associated with putrefaction or gangrene of the tissues, is indicative of infections with putrefactive bacteria.

Allied Processes.—Clinically there are a number of processes which are closely allied; sometimes the formation of gas being the most prominent feature of the clinical picture (gas phlegmon); at other times gangrene (noma, hospital gangrene).

Secondary Infections.—Secondary infections with pyogenic bacteria, most frequently the streptococcus, are common in these cases. The clinical picture, however, of putrefactive inflammation is always due to the putrefactive bacteria, even when there is a secondary infection. The general infection following the local infection is apparently often purely pyogenic in character, as streptococci are frequently found in the blood. It cannot be denied, however, that many of the severe general symptoms are due to the absorption of the toxic products elaborated by the putrefactive bacteria. The term pyosepthæmia was formerly applied to these mixed forms of general putrefactive and pyogenic infections; we will avoid the use of this term for reasons already given (p. 281). The character of the inflammation at the infection atrium is the best index as to the character of the general infection. If the local infection is of a putrefactive character, the general infection should be regarded as putrefactive, even if streptococci are found in the blood, as it is impossible to demonstrate the toxic products which are formed by the activity of putrefactive bacteria. If, however, the local inflammation is suppurative in character, the general infection should be regarded as pyogenic, even if putrefactive bacteria may be demonstrated in the local inflammatory focus.

The bacilli of the colon group form a peculiar intermediate group between the pyogenic and putrefactive bacteria; depending upon their action they are sometimes classed with the former, sometimes with the latter.

Relation between Putrefaction and Fermentation; Substances produced by Putrefaction.—Putrefaction is closely allied to fermentation; the former is a reduction, the latter an oxidation process. [Putrefaction may be roughly considered as a form of fermentation of albuminous and albuminoid matter. It seems that the first change is peptonization, effected in part by the peptonizing enzymes possessed by most organisms of decomposition. The peptonized substances are then further changed and split up; amido-derivatives (especially amido-acids), aromatic bodies, and sulpho-acids are next formed, and these are further split up, indol being among the final products.

The list of substances which may appear during putrefaction is very extensive; it contains among the gases CO_2, H, H_2S, and CH_4; among the fatty acids acetic, butyric, and valerianic acids, besides various amido-compounds, amins, indol, skatol; bodies of the aromatic series; various ptomains; basic compounds, etc., the chemical combinations varying qualitatively and quantitatively with the peculiar bacterial species

concerned and with the decomposing medium. The result of putrefaction in all cases is the decomposition of highly complex substances into others of simpler and more assimilable structure. It is especially under anærobic conditions that the odor of putrefaction is observed.—(Allbutt's *System of Medicine*, vol. i, p. 529.)]

There are formed in putrefaction, in addition to the metabolic products of the bacteria, complicated toxic and nontoxic substances, the so-called putrefactive alkaloids or ptomains (Selmi) and the toxic albuminous substances or toxalbumins.

Investigations as to the nature of the active substances in the putrefactive exudates were made long before the most important bacteria were discovered. Panum (1855) obtained a poisonous extract, von Bergmann and Schmiedberg (1868) obtained a crystalline body, sepsin, and were the first to suggest that it was to be regarded as the direct secretion product of bacteria, and not as a decomposition product of the albumins.

Nencki (1876) was the first to isolate a pure ptomain. Brieger (1885–86), employing improved chemical methods, isolated a number of nitrogenous bases from putrefying masses. To the poisonous ptomains, to which he gave the generic term toxins—a name now applied only to poisons secreted by living bacteria—belong peptotoxin, neurin, muscarin, etc., while other bases, such as neuridin, gadinin, putrescin, cadaverin, saprin, cholin, mydatoxin, mydin, are nontoxic, or only toxic when administered in large amounts.

The putrefactive have not been as thoroughly studied as pyogenic micro-organisms. This is partly due to the fact that putrefactive infections are rare at the present time, because of the methods employed in treating wounds, and partly to the fact that large numbers of saprophytes are deposited in necrotic tissue, and it is difficult to differentiate between these and the putrefactive bacteria.

Bacteria of Putrefaction.—The principal causes of putrefactive wound infections belong to the large group of bacteria, of which the proteus and colon bacilli, the bacillus aerogenes capsulatus, and the bacillus of malignant œdema are the most important.

Proteus Vulgaris.—Hauser described under the name of proteus vulgaris a form of bacillus which frequently occurs in putrefying animal tissues. This bacillus is from 1.2 to 4 μ in length, and has an average width of 0.6 μ. It is provided with numerous flagella, and is therefore actively motile; does not stain with anilin dyes, and only occasionally by Gram's method.

It is a facultative anærobe and grows upon ordinary culture media. If there is a free access of air gelatin is rapidly liquefied and foul-smelling substances, such as indol and sulphuretted hydrogen, are formed. When air is excluded the growth is much the same, but the

gelatin is then not liquefied. Gas is formed and an acid reaction develops in culture media containing grape-sugar; urea is transformed in carbonate of ammonia.

Gelatin cultures are most characteristic. Small depressions filled with white masses form rapidly upon the surface of the gelatin. From a turflike zone, which surrounds the depression resulting from liquefaction, delicate processes composed of bacilli, arranged in the shape of threads, run in all directions.

There is produced in culture media a poisonous alkaloid, which, according to E. Levy, is identical with the sepsin isolated by von Bergmann.

Subcutaneous injections of cultures into experimental animals produce abscesses; intravenous injections, toxic symptoms and metastatic foci of suppuration. The bacillus seems to be able to multiply in living tissues only when the latter have been injured by other bacteria or have become necrotic; the converse is also true, that the proteus favors the development of other varieties of bacteria; for example, streptococci and colon bacilli.

The bacillus is very widely distributed, even occurring in the fæces of healthy individuals, and it may easily be understood why it so frequently gains access to neglected wounds and ulcers.

In man the proteus vulgaris is found most frequently in foul-smelling gangrenous foci and gangrenous phlegmons. In rare cases they are the only bacilli to be found in the inflammatory focus, and may even invade the blood, and be deposited in the different viscera (Krogius). Usually, however, other bacteria, such as the colon bacilli and streptococci, are also found, and the clinical symptoms of the general infection are caused by the absorption of the toxic products of the proteus combined with the bacterial blood infection.

The proteus vulgaris occurs with the bacterium coli commune very frequently in decomposing meat (van Ermengem). When such meat is eaten the proteus is carried into the intestine, where it multiplies and elaborates its toxic products, causing diarrhœa, cholera nostrans and general toxic infections (poisoning by decomposed meat, sausage poisoning).

It also occurs with the colon bacillus in wounds, fistulæ, and ulcers about the anus, which become infected through the fæces. The proteus vulgaris may easily pass into the urethra and bladder, and even to the pelvis of the kidney, causing cystitis and pyelonephritis. These infections occur most frequently after catheterization. They may pass into the genital tract of the female, and are frequently found associated with streptococci in puerperal infections. They are found in peritonitis, associated with colon bacilli and pyogenic cocci, following intestinal perforation.

The proteus may pass from the intestines along the bile ducts to the gall bladder, and in rare cases may be carried by way of the blood to the different organs (strumitis, Tavel).

LITERATURE.—*Brunner.* Wundinfektion und Wundbehandlung. Frauenfeld, 1898, II and III.—*v. Ermengem.* Die pathog. Bakterien d. Fleischvergiftungen. In Kolle-Wassermanns Handb. d. pathog. Mikroorganismen, Bd. 2, 1903, p. 637.—*Hauser.* Ueber Fäulnisbakterien und deren Beziehungen zur Septikämie. Leipzig, 1885.—*Horn.* Ueber das Vorkommen des Proteus vulgaris bei jauchigen Eiterungen. I.-D., Erlangen, 1897.—*Kruse.* Bazillen. In Flügges Die Mikroorganismen, Leipzig, 1896.—*E. Levy.* Experim. und Klin. über die Sepsinvergiftung und ihren Zusammenhang mit Bact. proteus. Arch. f. exp. Path., Bd. 34, 1894, p. 342.

Bacilli of the Colon Group.—The bacilli of the colon group (Bacillus Coli Communis) have already been mentioned in discussing pyogenic bacteria, for they not infrequently produce local and metastatic inflammatory processes ending in pus formation. Frequently they are associated with the proteus vulgaris in wounds infected by fæces, in perforative peritonitis, and in poisoning following the ingestion of decomposing meat. Not infrequently they are found in gangrenous foci associated with pyogenic cocci. They have been found alone in cases of gas phlegmon.

In general infections they may be found in large numbers in the blood even during life. [No significance should be attached to the presence of colon bacilli in the blood shortly before or after death. It has been known for a long time that colon bacilli pass into the blood shortly before death, and that they are found in large numbers after death. The term agonal infection has been applied to the blood infections occurring at this time.] It is not known why in one case the colon bacillus produces pus and in another case foul-smelling gangrene, accompanied by gas formation. It apparently depends less upon the variety of the colon bacillus than upon the influence of symbiosis with other bacteria, especially the proteus, with which it is frequently associated in infections caused by fæcal matter.

Bacillus Emphysematosus.—The bacillus emphysematosus (bacillus aerogenes capsulatus) was found by E. Fränkel in four cases of gas phlegmon, three times associated with pyogenic bacteria, in one case alone, and accurately described by him in 1893. Rosenbach and E. Levy had seen the bacillus earlier than this; Welch and Nuttall had described it as the bacillus aerogenes capsulatus in 1892. It has been found in the intestinal canal of man and animals; in garden earth and the dust of the streets (Hirschberg).

It is an anærobic bacillus, resembling closely the anthrax bacillus, but forms no spores. It stains with anilin dyes and by Gram's method. It grows upon gelatin without liquefying it and without forming gas. In glycerin agar, to which sodium formate has been added, gas of the

odor of hydrogen sulphid is formed, while in glucose agar an odorless gas is produced.

Guinea-pigs develop after subcutaneous injections a nonsuppurative inflammation, ending in gangrene and accompanied by the formation of odorless gases. A foul-smelling purulent exudate develops as in the mixed infections occurring in man, when pyogenic cocci are injected at the same time.

LITERATURE.—*E. Fränkel.* Ueber Gasphlegmone, Schaumorgane und deren Erreger. Zeitschr. f. Hygiene, Bd. 40, 1902, p. 73.—*Passini.* Stud. über fäulniserregende anaerobe Bakterien des menschl. Darmes und ihre Bedeutung. Zeitschr. f. Hygiene, Bd. 49, 1905, p. 135.—*Stolz.* Die Gasphlegmone des Menschen. Beitr. z. klin. Chir., Bd. 33, 1902, p. 72.

Bacillus of Malignant Œdema.—The bacillus of malignant œdema is the cause of malignant œdema in man, and is therefore one of the most important of the group of bacilli producing a spreading inflammatory œdema associated with emphysema, and eventually ending in gangrene.

It is narrower than the anthrax bacillus and has rounded ends. In the tissues and in cultures it grows out into long filaments, which may be uniform throughout or segmented at irregular intervals. Under suitable conditions they form long spores which lie near the center of the rods.

The bacillus is a strong anærobe, and can therefore only be grown when air is excluded, but it can be grown without difficulty upon ordinary culture media, gelatin being liquefied and gas formed. Large amounts of foul-smelling gases are formed when the bacilli are grown in culture media containing grape-sugar, and in blood serum, for the bacilli belong to the bacteria which decompose albumins with the development of foul-smelling gases (Jensen). The bacilli, the spores, and the flagella may be stained by the methods ordinarily employed. The bacilli lose their color in Gram's method, in this way differing from those of anthrax.

The bacillus of malignant œdema is very widely distributed, being frequently found in decomposing fluids, in impure water, in the upper layers of the earth, down to 1 m. in the dust, in the intestinal canal and fæces of herbivorous animals, and therefore it occurs frequently in milk and farm produce. It has been demonstrated upon the human mucous membranes (Jensen).

It thrives especially well upon necrotic tissues and in deep wounds with pockets and recesses. It produces, following wound infections in horses or puerperal infections in cows, an acute febrile, frequently fatal, disease characterized by an extensive serohæmorrhagic and emphysematous œdema.

It was first described in man by Brieger and Ehrlich (1882) in two cases of rapidly progressive œdematous inflammation, which soon ended fatally. Feser and Pasteur had found the bacillus as early as 1876 in inflammations which they had produced in experimental animals by the injection of decomposing materials. The name was given it by Koch, who first described it accurately.

The toxins, which are negatively chemotactic, are formed in small amounts only. As the toxins are negatively chemotactic, there is no emigration of leucocytes. They irritate the blood vessels and nerves, causing an accumulation of a serous exudate.

LITERATURE.—*Jensen.* Malignes Oedem. In Kolle-Wassermanns Handb. d. path. Mikroorg., Bd. II, 1903, p. 619.

Conditions Essential for Putrefaction.—Two conditions are essential to the development of putrefactive changes: (1) The presence of degenerated or necrotic tissue. (2) Deep recesses or pockets in a wound to which the air does not have free access. Apparently symbiosis of the putrefactive bacteria with a number of different anærobic saprophytes plays an important rôle in the putrefactive infections. The virulence of putrefactive bacteria is increased by streptococci, and the putrefactive infections are more frequently mixed infections with the proteus and colon bacilli and streptococci than monoinfections. Tetanus bacilli find conditions favorable for development in wounds in which there are putrefactive changes.

Ectogenous Putrefactive Infections.—There are two reasons why ectogenous putrefactive infections are rare, notwithstanding the fact that the putrefactive bacteria are so widely distributed, and that surgeons are often compelled to work under adverse conditions, especially in war: (1) Putrefactive bacteria cannot develop in superficial accidental wounds because of the free access of air; (2) in the treatment of wounds as carried out at the present time all the infected recesses are opened and drained, and the dressings are so applied as to permit of a free access of air; and as a result putrefactive infections rarely develop even in compound fractures or machine injuries, which were infected at the time of the accident or later by meddlesome laymen. Severe ectogenous putrefactive infections occur almost only in injuries such as those described above in which the surgeon is called late. Putrefactive puerperal infections should be regarded as of ectogenous origin, as in the majority of cases the causes of the infection (colon and proteus bacilli) are carried into the vagina when proper aseptic precautions are not taken in making an examination.

Endogenous Putrefactive Infections.—Endogenous putrefactive infections developing from the mucous membranes of the mouth, intes-

tines, bladder, and urethra are more frequent than the ectogenous. These infections occurring in the mouth are associated with a number of lesions which lead to the formation of circumscribed and superficial areas of gangrene, occurring as secondary infections in stomatitis ulcerosa, angina gangrenosa, and pharyngeal diphtheria. They follow frequently the laceration of the tongue, produced by shooting into the mouth in attempted suicide. The putrefactive phlegmon which then develops in the floor of the mouth spreads along the layers of the cervical fascia and may cause an œdema of the glottis, which may prove fatal unless proper treatment is instituted. A retropharyngeal phlegmon follows an injury of the pharynx. A phlegmon about the œsophagus develops when a foreign body which has become arrested ulcerates through the œsophagus, when an œsophageal carcinoma ruptures externally, and after injuries produced by the œsophagoscope. Phlegmons developing from either the pharynx or œsophagus may extend between the layers of the cervical fascia to the mediastinum and cause death. Periostitis alveolaris leading to the formation of a foul-smelling, usually benign, abscess frequently develops from a periodontitis produced by a carious tooth with a decomposing pulp. Very rarely, however, does a fatal general infection follow the latter lesion. If a carcinoma of the tongue or larynx becomes infected with putrefactive bacteria, they may be carried to the lungs, and an inflammation, which is rapidly followed by gangrene, then develops. In gunshot wounds of the lungs gangrenous foci, putrefactive pleurisy, and secondary infection of a hæmothorax may be produced either by putrefactive bacteria which have been aspirated or by those which have been carried into the wound upon a piece of clothing. Phlegmons should also be mentioned which develop about the trachea secondary to carcinomatous, tuberculous, and syphilitic lesions of the larynx.

Putrefactive Peritonitis following Perforation and Rupture of the Intestines.—Perforation of the intestines is followed by an acute, progressive, putrefactive peritonitis, accompanied by a fatal general infection which is usually toxic in character, for in sudden perforations there is no agglutination of the intestinal loops, and the inflammatory process is not walled off; therefore the intestinal ferments and bacterial toxins which destroy the protecting endothelium and the bacteria (especially the proteus and colon bacilli associated with different pyogenic cocci) are poured directly into the free peritoneal cavity.

Periprocteal Abscesses and Infections following Extravasation of Urine.—A benign circumscribed phlegmon (the periprocteal abscess) from which fistulæ in ano develop frequently forms about the rectum following injuries by inspissated fæcal masses. Injuries of the bladder and urethra are frequently followed by severe phlegmons when the ex-

travasated urine contains putrefactive bacteria. Mild endogenous putrefactive infections are occasionally seen after operations about the rectum, and in operations upon the intestines when the fæces are permitted to flow over the wound. Similar infections occur in plastic operations upon the lips and cheeks when a poorly nourished flap dies and becomes gangrenous following infection with bacteria from the mouth cavity.

Symptoms of Putrefactive Infections.—When putrefactive inflammation develops in a wound the temperature either rises abruptly with a chill or steadily, and the appearance of the wound becomes very characteristic within twenty-four hours. A foul, repellent odor arises from the dry wound surfaces. The loosely attached shreds of tissue and the skin edges for a varying distance have a bluish or black color. Twenty-four hours later small amounts of a brownish or greenish ichorous discharge are poured out from the deepest recesses of the wound, while the inflammatory redness and swelling, accompanied by an increasing pain, extend into the surrounding tissues. Soon the wound surfaces become discolored and moist, and the large shreds of necrotic tissue become liquefied or are cast off. When pressure is made upon the surrounding tissues, gas bubbles appear in the ichorous discharge. If the inflammatory process subsides the inflammatory swelling and ichorous discharge gradually disappear, healthy granulation tissue develops around the gangrenous area and healing occurs. In the worst cases a high continuous fever persists and the symptoms of general infection, which correspond exactly to those occurring in general pyogenic infections, develop; death occurring in a few days, frequently at the end of the first day. Frequently streptococci, more rarely staphylococci or colon bacilli, are found in the circulating blood. Even the proteus vulgaris has been found in the blood in these cases (Krogius). In most cases, however, bacteria cannot be demonstrated in the blood, which is laden with toxic materials as indicated by the severe changes occurring in it. If metastases develop, they have the same putrefactive character as the primary focus.

Putrefactive Phlegmon.—If the putrefactive inflammation extends, a phlegmon forms which develops more rapidly, and is accompanied by severer local and general symptoms than the most malignant streptococcic infections. The names which have been applied to putrefactive phlegmons by different authors, acute suppurative œdema ending in gangrene (Pirogoff), progressive gangrenous emphysema, emphysematous gangrene, fulminating gangrene (Maissoneuve), gangrenous emphysema, gangrenous phlegmon, panphlegmon gangrænosa (Fischer), etc., describe very well the clinical picture. Often within twenty-four hours an entire extremity becomes so œdematous and red that the painful swollen lymph nodes can no longer be palpated, and the red streaks indicative of lymphangitis, which is rarely absent, can no longer be

seen. The inflammatory exudate is so great that the circumference of the extremity becomes three or more times as great as normal, and exerts so much pressure that the circulation is interfered with. The tense skin becomes pale and anæsthetic, and large blebs with serohæmorrhagic contents form; bluish discolored areas develop and become transformed into black crusts, which are cast off as the gangrene extends. Fluctuation cannot be elicited anywhere in the enormously swollen extremity, but when the tissues are palpated an emphysematous crackling, which is characteristic of gas in the tissues, may be elicited. The fingers or toes are of a pale or bluish color and feel cold. When these infections develop from wounds they rarely remain confined to the subcutaneous tissues, but extend to the muscles and periosteum, and when a compound fracture becomes infected, the bone marrow is transformed into a decomposing, putrefying mass (putrefactive osteomyelitis). Putrefactive arthritis is accompanied by great destruction, the capsule becomes necrotic, the cartilages are separated and destroyed.

Appearance of Tissues when Incised.—When incisions are made the frightful results of these infections are revealed. The incision passes through an œdematous or gangrenous cutis into an œdematous, grayish-green subcutaneous tissue from which a sanious discharge containing gas bubbles and shreds of fat and fascia pour out. The intermuscular connective tissues and muscles are in the same condition. Everywhere a gelatinous, discolored network of tissue is found from which an ichorous discharge and gas bubbles can be expressed. The periosteum is raised from the bone and separated into gangrenous shreds, while from the bone marrow decomposing matter is discharged from the seat of fracture or through large canals in the bone. The subcutaneous and deeper veins become thrombosed, the thrombi frequently undergoing septic softening; the walls of the larger arteries have a grayish color and are about to rupture (gangrenous arteritis). Fatal hæmorrhage may follow the erosion of an artery by the gangrenous process.

Putrefactive Phlegmons following Urinary Extravasation, and the Escape of Fæcal Matter.—Putrefactive phlegmons develop in the scrotum, penis, and perineum following urinary extravasation. The urine is poured out into the tissues after injuries or inflammatory processes which destroy the integrity of the urinary passages, such as fractures of the pelvic bones, periurethritis following urethral strictures, and injuries produced while introducing catheters. A rapidly progressive swelling and redness, accompanied by severe pain and fever, indicate the beginning of the inflammation which leads to extensive gangrene of the muscles, fascia, and skin, if death from a general toxic infection does not occur before gangrene has time to develop. The greater the number of bacteria in the urine such as occur in cystitis following hypertrophy of

the prostate and urethral stricture, the more rapid and severe the inflammation will be.

A similar but less frightful clinical picture, as in this case pressure is not exerted as when urine is extravasated, is produced by the discharge of fæcal matter into the tissues, following, for example, gangrene of a strangulated intestinal loop in a hernia (fæcal phlegmon and abscess). Necrotic and dying tissue affords the best culture media for putrefactive bacteria, and the development of putrefactive inflammation following the various forms of necrosis is not at all rare unless prophylactic measures are instituted early. Bed sores about the sacrum and coccyx easily become infected from fæcal matter. Gangrene, and in neglected cases putrefactive phlegmons and general infections, may then develop. A similar infection followed by similar results not infrequently develops in tuberculous fistulæ about the perineum treated by quacks.

Putrefaction in Senile and Arteriosclerotic Gangrene.—Senile gangrene and arteriosclerotic gangrene of the fingers and toes readily pass from the condition of dry necrosis or mummification into that of a moist putrefactive gangrene, which affords opportunities for the development of phlegmons. A gangrene due to freezing, embolism of the arteries of the extremities, nervous lesions, and carbolic-acid compresses may also give rise to putrefactive phlegmons. Pyogenic and putrefactive infections develop most rapidly in diabetic gangrene. The resistance of the tissues is so reduced in diabetes that lymphangitis and phlegmons develop immediately after infection with putrefactive bacteria.

Treatment.—Prophylaxis is the most essential factor in the treatment of putrefactive infections. The detached and contused tissues should be removed from lacerated wounds, the edges of the wound should be trimmed off, then the wound should be loosely tamponed and should be drained and treated by the open method. Secondary infection should be prevented by sterilizing the surrounding area and avoiding any useless examinations or manipulations. If gangrene has already developed, a dry aseptic dressing should be applied. Putrefactive processes rapidly develop, especially in diabetic patients, when moist dressings are used. If the inflammation has already developed, the infected tissues should be freely exposed by opening the recesses and pockets of the wound and by incising freely the phlegmon or abscess. In the beginning a tampon of dry aseptic gauze should be used. Iodoform gauze is contraindicated, as the iodoform is quickly decomposed in putrefactive processes and may give rise to severe toxic symptoms. When an extensive gangrene has developed in the wound, or there is a superficial gangrene of the skin, compresses of a three per cent solution of acetate of aluminum, of boric acid or hydrogen peroxid solution may be used to hasten the separation of the dead tissue. When severe general symptoms de-

velop amputation of the inflamed extremity should be considered, although only in rare cases is one able to prevent the dangers of general infection even by this radical procedure.

(b) ALLIED PROCESSES

Gas Phlegmon.—The gas phlegmon may be called a variety of gangrenous phlegmon, which is characterized by the formation of large amounts of gas. The skin is raised from the subjacent tissue by large accumulations of gas, resembling an air cushion; while after death there is a rapid, progressive formation of gas in the viscera (so-called foam organs). The more pronounced the gangrene, the less marked are the inflammatory symptoms.

The bacillus aerogenes capsulatus and allied anærobic bacteria (butyric acid bacilli) appear to be the most important causes of gas phlegmon (Welch and Flexner, Muscatello, Hitzmann and Lindenthal, Stolz, Koprac). The bacillus aerogenes capsulatus was found by Lenhartz in the blood during life in a puerperal infection which ended fatally.

The proteus bacillus (Graszberger, Widal, and Nobécourt) and the bacterium coli commune (Chiari, Klemm, Bunge, Tavel, and others) are occasionally found in gas phlegmons.

Mixed infections with pyogenic bacteria are also frequent.

Malignant Œdema.—Malignant œdema is a term often applied to acute suppurative and putrefactive phlegmons; it should be used, however, only to designate those rare, rapidly progressive phlegmons accompanied by gas formation and gangrene of the skin and subjacent tissues, which are caused by the bacillus of malignant œdema alone or associated with other bacteria. Even after a bacteriological examination it is frequently impossible to make a positive diagnosis because of the similarity of the bacteria (Ghon and Sachs) found in these analogous inflammatory processes.

The exudate before gangrene begins is serous in character and contains but few cells. This is due to the fact that the bacterial toxins exert a negative chemotaxis; the same occurring in gas phlegmons, in which there is no secondary infection with pyogenic bacteria.

The treatment of gas phlegmons and of malignant œdema is the same as that of putrefactive phlegmons.

LITERATURE.—*Albrecht.* Ueber Infektionen mit gasbildenden Bakt. Arch. f. klin. Chir., Bd. 67, 1902, p. 514.—*E. v. Bergmann.* Zur Lehre von der putriden Intoxikation. Deutsche Zeitschr. f. Chir., Bd. 1, 1872, p. 373.—*Brieger.* Untersuchungen über Ptomaine, Berlin, 1885–6.—*Brunner.* Wundinfektion und Wundbehandlung. Frauenfeld, 1898.—*E. Fränkel.* Ueber Gasphlegmonen, Schaumorgane und deren

Erreger. Zeitschr. f. Hygiene, Bd. 40, 1902, p. 73.—*Ghon* und *Sachs*. Beiträgezur Kenntnis der anaeroben Bakterien des Menschen. Zur Aetiologie des Gasbrandes. Zentralbl. f. Bakt., 1903, Bd. 34, Orig., p. 289 and Bd. 36, Orig., 1904, p. 178.—*Kamen*. Zur Aetiologie der Gasphlegmone, Ebenda, Bd. 35, 1904, p. 554.—*Koprac*. Ein Beitrag zur weiteren Differenzierung der Gangrène foudroyante. Arch. f. klin. Chir., Bd. 72, 1904, p. 111.—*Lenhartz*. Die septischen Erkrankungen. Wien, 1903.—*Panum*. Das putride Gift, etc. Virchows Arch., Bd. 60, 1874, p. 301.—*Pirogoff*. Grundzüge der allgemeinen Kriegschirurgie. Leipzig, 1864.—*Sandler*. Ueber Gasgangrän u. Schaumorgane. Mitteilung u. Sammelreferat. Zentralbl. f. allgem. Path., 1902, p. 471.—*Stolz*.—Die Gasphlegmone d. Menschen. Beitr. z. klin. Chir., Bd. 33, 1902, p. 72.—*Westenhoeffer*. Weit. Beiträge z. Frage der Schaumorgane u. der Gangrène foudr. Virchows Archiv, Bd. 170, 1902.

Noma[1] (*Water Cancer, Gangrene of the Cheek*).—Noma is closely allied to those putrefactive infections in which gangrene predominates. It involves most frequently the cheeks, more rarely the gums, palate, and lips. Similar infections occur about the anus and vulva.

Etiology.—The disease attacks almost exclusively weak and emaciated young children from two to twelve years of age, living in squalid, over-populated districts in cities; much more rarely adults. Measles and typhoid fever, syphilis, mercurial stomatitis, diphtheria, dysentery, different forms of ulcerative stomatitis, malaria, poor hygienic conditions are predisposing and accessory causes which reduce the local and general resistance and prepare the tissues for the development of bacteria. Noma is apparently a bacterial infection, but a specific organism has not as yet been demonstrated.

As a rule, only isolated cases occur, and the possibility of direct transference from one patient to another can be excluded. Wherever a number of cases have developed in the same hospital or district, there has been a preceding epidemic of measles.

Bacteria and Fungi found in Noma.—A number of different bacteria and fungi have been found in noma. This is not to be wondered at when one considers the great variety and number of bacteria normally present in the mouth and found in putrefactive processes. Perthes demonstrated microscopically a streptothrix in the margin of the gangrenous area which sends out its terminal processes in the form of spirilla into the adjacent healthy tissues. He was unable to produce the disease in animals with this streptothrix or to grow it in pure cultures. Freymuth and Petruschky found the diphtheria bacillus in two cases of noma observed by them. It is doubtful, however, whether the diphtheria bacillus should be regarded as the cause of these cases of noma, as they are found in the mouth of healthy individuals. It is a question whether noma is caused by any single variety of bacteria, or whether it is caused by a number of different varieties (Kolle and Hetsch).

[1] From the Greek νέμεσθαι—to destroy.

Clinical Course.—The specific process begins in an abrasion or an inflamed area (ulcerative or mercurial stomatitis) in the mucous membrane of the cheek, near the angle of the mouth, occasionally in the mucous membrane of the palate, lips, or gums. Gangrene gradually develops from a vesicle with cloudy contents or a superficial ulcer, and extends both superficially and deeply. As the gangrene spreads the surrounding tissues become inflamed, indurated, and hard. A high fever develops and persists; the severity of the disease is indicated by the height of the fever and the mental disturbances. A swelling of the cheek which is not very painful develops, and soon there appears upon the pallid skin a bluish-black discoloration corresponding approximately to the area of mucous membrane which is infiltrated. This bluish-black discoloration of the tissues is characteristic of noma.

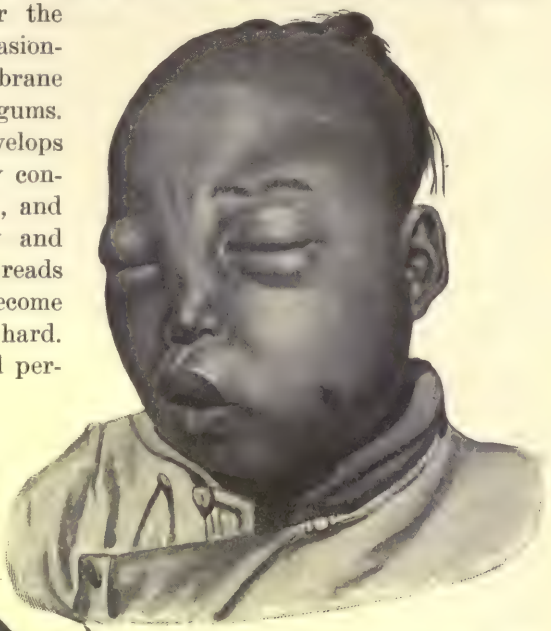

FIG. 127.—NOMA IN A CHINAMAN SIXTEEN YEARS OF AGE. (After photographs and communication of Professor Perthes.) Tenth day of the disease. The dark area in cheek is the point at which perforation is about to occur.

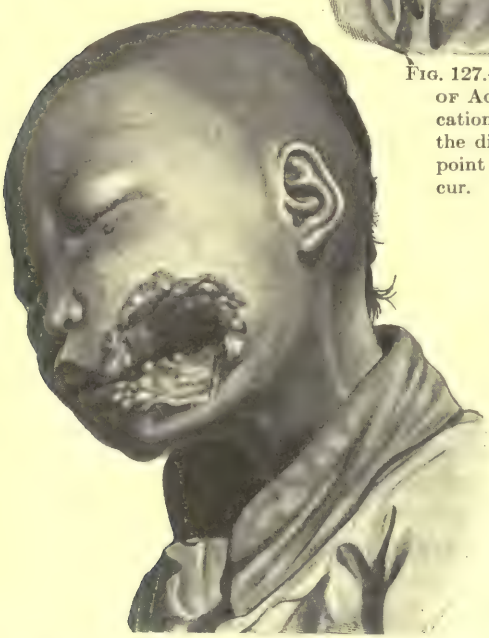

FIG. 128.—PICTURE TAKEN ON SIXTEENTH DAY OF THE DISEASE AFTER CAUTERIZATION. Death on twenty-second day, the disease having extended to the pharynx and soft palate.

As the disease progresses the inflammatory reaction may become marked and the entire face and the side of the neck becomes swollen, but the gangrenous process does not sub-

side. Within the first week the dark, gangrenous tissues slough out; the process extends and destroys the cheek, the mucous membrane covering the upper and lower jaws; the teeth become loosened and drop out, and the surfaces of the maxilla and mandible become exposed. In the malignant cases the gangrene extends to the nose, the tongue, the pharynx, the palate, the lips, and the other cheek. The amount of saliva is increased and a foul-smelling discharge is poured into the mouth, which may be swallowed and aspirated, causing gastrointestinal disturbances, bronchopneumonia, and gangrene of the lung.

Prognosis.—Death, which frequently occurs at the end of the first week, ends the frightful suffering in seventy-five per cent of the cases. It is due to paralysis of the heart, resulting from a general toxic infection, exhaustion, or pneumonia. In rare cases the gangrene subsides after the necrotic tissues have sloughed out, and then it does not extend into the surrounding inflamed tissues; healthy granulations form, the necrotic soft tissues and bone are separated and cast off, and healing occurs. Large defects of the cheek, lip, and nose, with cicatricial lockjaw and ectropion, remain after healing.

Fig. 129.—Deformity Following a Noma of the Face, Which Healed.

Treatment.—The treatment consists of destruction of the gangrenous tissue with the Paquelin cautery. The entire thickness of the cheek in which the gangrene develops should be cauterized, and even the healthy tissue immediately adjacent to it. Trendelenburg recommends splitting the cheek in order to expose more thoroughly the diseased area. Attempts have been made to prevent the putrefactive decomposition of the gangrenous tissue by the use of caustics (zinc chlorid, acetic acid) and to control the inflammation by using a five per cent solution of hydrogen peroxid as a mouth wash frequently. If treatment is instituted early, procedures which are not mutilating may be successful. Every effort should be made to improve the general condition of the patient.

It may be necessary to perform plastic operations to close the defects,

and to remove the masses of cicatricial tissue to cure the cicatricial lockjaw.

LITERATURE.—*v. Bergmann.* Verletzungen und Erkrankungen der Mundhöhle Handb. d. prakt. Chir., 2d Edition.—*Kolle* und *Hetsch.* Noma. In Kolle-Wassermanns Handb. d. pathog. Mikroorg., Bd. 3, 1903, p. 904.—*Krahn.* Ein Beitrag zur Aetiologie der Noma. Mitteil. a. d. Grenzgeb., Bd. 6.—*Perthes.* Ueber Noma und ihren Erreger. Chir.-Kongr. Verhandl., 1899, II, p. 63.—*v. Ranke.* Zur patholog. Anatomie des nomatösen Brandes. Münch. med. Wochenschr., 1903, p. 13.

Hospital Gangrene (*Wound Phagedæna*).—By hospital gangrene is understood a wound infection resulting in an acute progressive necrosis of the tissues with putrefactive decomposition of the same. The name of hospital gangrene has been given to this form of infection, as in preantiseptic times it frequently occurred in epidemic form in civil and military hospitals. The infection was transferred from wound to wound by the lint and sponges (the materials used for dressing wounds in earlier times), the instruments, and the fingers of the operator or his assistants. It attacked recent and old, large and small wounds, and pursued a rapid and severe course, which often ended fatally.

At the present time the surgeon occasionally sees a case of the milder form of hospital gangrene, which develops most frequently about the anus or the external genitalia. Operation- and accidental-wounds are but rarely attacked by this form of infection.

Etiology.—Hospital gangrene is apparently of bacterial origin, but no specific bacterium has yet been found. Vincent and Matzenauer have demonstrated a bacillus in a number of cases, but have been unable to grow it in pure cultures. Nasse found in one case an amœboid organism.

If one reads the descriptions of hospital gangrene given by the older authors, one cannot help thinking that a number of different infections —putrefactive gangrene and phlegmon, perhaps even gas phlegmon, and infections with the diphtheria bacillus (wound diphtheria)—were grouped under this term.

Clinical Course and Forms.—Depending upon the course, authors have differentiated a superficial and a deep form of hospital gangrene (Phagedæna superficialis et profunda, von Pitha, König); upon the gross appearance an ulcerative and a pulpy gangrene (Delpech).

The symptoms begin after an incubation period of from two to three days with fever, pain, and changes in the appearance of the wound. In the ulcerative form which attacks especially granulating wounds, the wound surface becomes mottled with yellowish-brown areas, and small hæmorrhages occur within the granulations. If the changes are mild and not progressive, one speaks of a diphtheritic form of hospital gangrene. In the ulcerative form the gangrene extends rapidly over the entire wound, transforming the tissues into a discolored, foul-smelling mass

When the gangrenous tissues are cast off, ulcers with sharply cut edges remain which rapidly coalesce. While the hæmorrhagic mottled floor of the ulcer becomes gangrenous, the sharply cut, irregular borders of the ulcer, surrounded by painful, inflamed, and infiltrated skin, extend. The changes characteristic of the pulpy form of gangrene may develop in the floor of the ulcer. The floor of the wound then becomes dry and covered with a thick, dirty, fibrinous membrane, which may be removed in shreds, leaving bleeding surfaces. After a short time an ichorous secretion is poured out, and the surface of the wound, as the result of putrefactive changes and the development of gas within the tissues, becomes transformed into a grayish black or yellowish gray, firmly attached, semifluid mass (so-called pulp), which has been compared to decomposing brain matter. These changes, which may frequently be associated with considerable parenchymatous hæmorrhage (hæmorrhagic form), may develop in a single night in large, recent operation-wounds (for example, after an amputation).

If the process remains superficial, the surface of the wound may gradually become clean, and covered with healthy granulation tissue, but the infection may recur at almost any time until healing is complete.

In the more malignant forms, the process, which resembles closely a putrid phlegmon, extends deeply, involving the loose subcutaneous and intermuscular tissues and the connective tissues of the vascular sheaths. If the resistant fasciæ are destroyed, large pieces of decomposing muscle are extruded. If the process still extends the periosteum is destroyed and the superficial layers of the bone become necrotic, the walls of the vessels ulcerate, and fatal hæmorrhages may occur.

Prognosis.—The dry forms of hospital gangrene may cause death within two days by rapid extension, accompanied by a general toxic infection. The mortality, depending upon the hygienic conditions and the simultaneous occurrence of other diseases (cholera, typhoid and dysentery), differs. It varies, according to the statistics compiled by different authors, from 6 to 80.6 per cent (König).

Character of the Fever.—The fever may be continuous or remittent, and may fall abruptly when the gangrene subsides, and after the use of the actual cautery or caustics. In rare cases it begins with chills.

The general symptoms correspond to those developing in general pyogenic and putrefactive infections.

Complications.—Erysipelas, metastatic suppuration, lymphangitis, lymphadenitis, and mixed infections with pyogenic bacteria are the most frequent complications.

Diagnosis.—The diagnosis of hospital gangrene under conditions existing at the present time is difficult. So few cases are seen that one does not have enough clinical experience to enable him to recognize the

milder forms, and the severer forms of hospital gangrene are not seen at present. It is scarcely possible to differentiate wound diphtheria from the milder forms except by microscopic examination unless the infection develops in a wound upon a patient already suffering with diphtheria.

Treatment: Prophylactic and Operative.—The present method of treating wounds, and improved hygiene in both civil and military hospitals prevent the development of the infection.

Complete isolation of patients suffering with hospital gangrene is not necessary. They should be separated from patients recently operated upon with clean wounds, but may be kept without danger in wards in which patients with pyogenic and other infections, erysipelas, etc., are isolated.

The early and energetic use of caustics, concentrated zinc chlorid solution, and nitric acid, or at the present time the actual cautery, plays an important part in the treatment. Deep-lying gangrenous foci should be exposed by incisions and rendered accessible.

If the hæmorrhage is severe, the principal artery supplying the part should be ligated at some distance from the gangrenous area, in healthy tissues at the point of election.

Amputation is indicated when the general symptoms become severe, and may be the only measure which will save the life of the patient.

LITERATURE.—*Delpech.* Mémoire sur la complication des plaies et des ulcères connues sous le nom de pourriture d'hôpital. Paris, 1815.—*v. Heine.* Der Hospitalbrand. Handb. d. Chir. v. Pitha-Billroth, Bd. 1, 2. Abt., 1869–74.—*König.* Ueber Hospitalbrand. v. Volkmanns Samml. klin. Vortr., No. 40, 1872.—*E. Küster.* Hospitalbrand. In Eulenburgs Realenzyklopädie.—*Matzenauer.* Zur Kenntnis und Aetiologie des Hospitalbrandes. Arch. f. Dermat. u. Syphil., Bd. 55, 1901, p. 394.—*Nasse.* Ueber einen Amöbenbefund bei Leberabszessen, Dysenterie und Nosokomialgangrän. Arch. f. klin. Chir., Bd. 43, 1892, p. 40.—*Rosenbach.* Der Hospitalbrand. Deutsche Chirurgie. Lief. 6, 1889.

CHAPTER VII

SUPPLEMENT TO THE TREATMENT OF ACUTE INFLAMMATION

THE recognition of the fact that local infections (pyogenic and putrefactive) could not be reached by drugs administered or applied externally, resulted in the establishment of the fundamental principle of early incision to permit of the escape of infectious material, and the use of the tampon, which removed these materials by its capillarity.

Bier in 1905 introduced still another method by which it may be pos-

sible to combat infections. In the Bier treatment an attempt is made to increase the natural resistance of the tissues by inducing a local passive hyperæmia, and to place the organism in a condition in which it can resist infection, and by avoiding large incisions, immobilizing dressings, and tampons, and by early motion to restore the function of the diseased extremity.

But such a method, in which the greater part of the struggle against infection is left to the organism, can be employed with safety only when the infection is mild. It is doubtful whether the treatment will suffice in severe infections, for in these more than in any other the final results depend upon the resistance of the organism, which only rarely can be estimated in the beginning of an infection.

Passive hyperæmia, according to Bier, is induced by the application to the extremity involved of a thin elastic constrictor 6 cm. in width. The constrictor is applied near the trunk over a few turns of a gauze bandage or about the neck under slight tension. The constrictor is fastened with a safety pin, or, according to Klapp, by cohesion of the ends of the constrictor after having been placed in water.

The constrictor may be applied at higher or lower levels several times during the day. A piece of rubber tubing may be used above the shoulder and about the testicle, and an elastic bandage 3 cm. in width about the neck.

The constrictor should not be applied tight enough to stop the circulation or even to weaken the pulse, the object being merely to slow the blood stream and to cause a dilatation of the blood vessels. When properly applied the extremity becomes hot and œdematous, and the pain in the inflammatory area rapidly subsides. If the constrictor is too tight the pain increases in severity. It is difficult to maintain the proper degree of hyperæmia, and the patient must be continually watched, especially if not very intelligent, as it may be necessary to remove and reapply or to readjust the constrictor several times during a treatment. Many have difficulty in maintaining a warm hyperæmia with an acute œdema. If the constrictor exerts too great compression the nutritional disturbance resulting from the increased stasis injures the tissues and reduces their natural resistance.

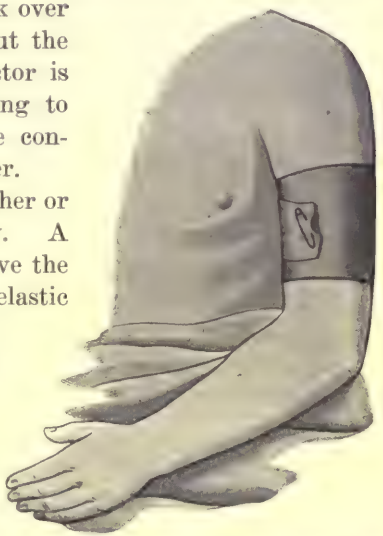

Fig. 130.—Band for Producing Passive Hyperæmia Applied to the Arm. (After Bier.)

The constrictor in the beginning, applied daily, may be allowed to re-

main ten hours, later as long as twenty-two hours. When it is removed the extremity is elevated or suspended in order to lessen the œdema.

Small punctures may be made into the inflamed area to assist in the treatment. Large incisions are employed only when there are severe circulatory disturbances in the inflamed area. Incisions are made when softening has occurred and there is an accumulation of pus. Tampons are not used, however, as the transudate which is poured out in such large amounts following the passive hyperæmia keeps the wound open.

Only deep wounds are drained. The pus is expressed each day when the dressings are changed. From the first day active and passive motion is begun (even when there is an inflammation of tendon sheaths or a joint), the object being to obtain as good functional results as possible. For the same reason no immobilizing dressing is applied, the wound being merely covered with several layers of gauze, which are maintained in position by a loosely applied roller bandage.

The method has been recommended for the treatment of all kinds of acute inflammatory processes, especially of a pyogenic character, of the extremities, head, and testicle (lymphangitis, phlegmons of all kinds, felons, suppuration of bones and joints, gonorrheal arthritis, infected open accidental- and operation-wounds), and to hasten the separation of necrotic tissues, etc. Although the method has been enthusiastically received, there are some serious objections to it. In private practice it is not entirely practical, for the patient must be watched continually.

According to Lexer's experience good results may be obtained in mild infections which do not progress rapidly, and are accompanied by little or no fever if the treatment is instituted during the first few days. These infections, however, subside completely or end in the formation of a small abscess, which rapidly heals when a small incision is made, just as frequently when an immobilizing dressing is applied and moist compresses are used. Often, however, under this treatment, the inflammatory infiltrate increases in size (even in mild cases in which a hyperæmia has been early induced), and there develops still more rapidly than when poultices, which are no longer used to-day, are employed an acute, rapidly progressive phlegmon which ruptures into and invades the healthy surrounding tissues. The local inflammation becomes worse and extends.

Phlegmons of the tendon sheaths and suppurative arthritis heal with good functional results if the hyperæmia is induced early. It is well known that good functional results have been obtained in these cases by the usual treatment, but good functional results are much more frequent when Bier's method is employed, and the clinical course is shorter than when early incisions and dry dressings are used, but the treatment must be continued until the inflammation has completely subsided in order to prevent, with certainty, recurrences.

In some of the severe cases the local and general condition has become worse even when the hyperæmia has been induced early. I am convinced that some of the bad results that I have seen follow this treatment could have been avoided if immediate, early incision combined with the use of a tampon had been employed. Some of the bad results which I have had may be briefly mentioned: (1) Rapid extension of the inflammation with the formation of a large inflammatory infiltration and abscesses; (2) rupture of the abscesses into healthy surrounding tissues; (3) general infection in a streptococcic arthritis of average severity upon which the treatment had a favorable influence for one week; (4) a fatal case in a child with a pneumococcic infection of the knee joint. In the last case two days after the hyperæmia was induced the temperature, which had been 101° F., rose to over 104° F. with symptoms of severe intoxication, and death rapidly followed.

The most effective factor in the treatment is not the bacteriolysis produced by an accumulation of the protective substances, which may set free a large amount of endotoxins injuring the tissues and the organism; not the dilution of the toxins by the œdema, nor the increased absorption which follows the removal of the constrictor; but the mechanical flushing and washing of the inflamed and œdematous tissues by the greatly increased amounts of transudate.

Recent large, open, accidental-wounds are cleansed very quickly and heal without infection if an hyperæmia is induced. It may be used to advantage in the treatment of inflammatory infiltrations before they have softened, when combined with large enough incisions to permit of the escape of the transudate. Passive hyperæmia acts favorably in severe cases, and a tampon can be dispensed with if large incisions, which permit of the escape of the infectious materials, are made before the hyperæmia is induced. If incisions are not made the hyperæmia may do harm, for the transudate, like Schleich's solution when used in acute inflammations, may drive the bacteria and their toxins into healthy tissues and favor the extension of the inflammation.

Therefore, in my opinion incisions should not be delayed until softening has occurred, but should be made, especially when there is an acute febrile onset, before the hyperæmia is induced. Cavities containing pus should be opened wide in order to permit of a free discharge of the pus and the transudate which follows the application of the constrictor. Hæmorrhage must naturally be controlled by a tampon before the hyperæmia is induced, the transudate favoring the separation of the gauze.

Expression of the pus by digital pressure, the avoidance of immobilizing dressings, and early movement of the diseased extremity are not to be recommended. Passive hyperæmia is not to be recommended in the treatment of acute lymphangitis.

Klapp has recommended for the treatment of small inflammatory foci, especially those occurring upon the trunk, an apparatus from which the air may be exhausted which resembles somewhat the dry cup formerly employed very extensively. Naturally the pressure exerted by the edges of the glass should be removed from the acutely inflamed area.

FIG. 131.—SUCTION APPARATUS FOR MASTITIS. (After Klapp.)

The suction glasses (Fig. 131), which are made in different forms and sizes, should be applied daily for about three quarters of an hour in all. After being in position for five minutes the glass should be removed for from one to three minutes and then reapplied. The apparatus should be boiled before using, and vaseline should be applied to

FIG. 132.—SUCTION APPARATUS FOR FELONS. (After Klapp.)

the skin where the rim comes in contact in order to prevent the glass from falling off. The vacuum should not be too low, and the glass should not be allowed to remain in position too long, as hæmorrhages may occur into the tissues.

Furuncles and carbuncles with necrotic centers, small inflammatory foci on the fingers, in the breast and lymph nodes, which have already

softened and have been opened by a small incision, suppurating hæmatomas—in short, all encapsulated foci not accompanied by fever and without a tendency to progress—may be quickly rid of pus and infectious materials by this suction treatment, without any added injury to the tissues. Large incisions may be avoided in this way and the time required for healing shortened. Beginning mild inflammations, so common upon the hands of physicians, may be easily controlled. Cautious treatment with a suction apparatus is to be recommended for all cases of this character. According to my experience, however, it should not be recommended for the treatment of inflammatory infiltrations which

FIG. 133.—SUCTION GLASS FOR A FURUNCLE. (After Klapp.)

have not softened and which are accompanied by fever and have a tendency to extend rapidly. An inflammation of this character, which after early incision and use of the dry tampon subsides in from one to two days with an immediate decline in the temperature, may extend and be associated with the formation of large amounts of pus and an extensive destruction of tissue when this treatment is employed. The local condition is aggravated and the time required for healing is lengthened, and even after the inflammation has subsided the induration persists and is more extensive than when early incisions, not combined with the suction treatment, are made.

LITERATURE.—*Bestelmeyer.* Erfahrungen über die Behandlung akut entzündl. Prozesse mit Stauungshyperämie. Münchn. med. Wochenschr., 1906, p. 461.—*Bier.* Behandlung akuter Eiterungen mit Stauungshyperämie. Ibid., 1905, p. 201;—Die Hyperämie als Heilmittel. Leipzig, Vogel.—*Klapp.* Ueber die Behandlung entzündl. Erkrankungen mittelst Saugapparaten. Münchn. med. Wochenschr., 1905, p. 740.—*Lexer.* Zur Behandlung akuter Entzündungen mittelst Stauungshyperämie. *Ibid.*, 1906, No. 14;—Die Behandlung der septischen Infektion. Zeitschr. f. ärztl., Fortbildung, 1906.—*Ranzi.* Ueber die Behandlung akuter Eiterungen mit Stauungshyperämie. Wiener klin. Wochenschr., 1906, No. 4.—*Wolf-Eisner.* Die Biersche Stauungshyperämie vom Standpunkt der Endoxinlehre. Münch. med. Wochenschr., 1906, p. 1102.—[Siehe auch die Diskussion über das Thema in den Chir.-Kongr.-Verhandl., 1906.]

CHAPTER VIII

SURGICAL HÆMATOLOGY

HÆMATOLOGY, though but recently introduced into the fields of diagnosis and prognosis, has given results of such great value that a very brief consideration of its more important branches as related to surgery is here presented. A more systematic study of the various changes of the blood in many surgical conditions will not only lead to more accurate diagnoses and methods of treatment, but will help to clear up many of the obscure problems in connection with the pathogenesis of disease. Much has been written recently upon the value of blood examinations. No one questions the great value of a positive blood culture or other results equally decisive. Negative findings and those changes which are not so constant—e. g., the presence or absence of a leucocytosis—have led to widely different conclusions. This much is certain, however, that in the hands of competent men the examination of the blood, when correlated with the clinical symptoms, will lead to the identification of many puzzling conditions.

For the technical methods involved, the reader is referred to works on laboratory diagnosis and hæmatology.

BACTERIOLOGY OF THE BLOOD

The bacteriological examination of the blood is frequently the means of identifying many puzzling septic conditions. Many descriptions given previously convey erroneous ideas as to the technic involved and as to the interpretation of the results. The statements frequently made that the demonstration of streptococci in the blood in cases of septic infections means a fatal issue, and that the presence of pneumococci and typhoid bacilli in the blood in cases of lobar pneumonia and typhoid fever respectively is to be regarded as a very bad prognostic sign are erroneous. These statements are usually based either upon insufficient data or upon results obtained by a crude technic. It can readily be understood how the demonstration of a bacteræmia by methods wholly inadequate—unless the bacterium sought for is present in very large numbers, as occurs frequently in overwhelming infections—will lead to the erroneous conclusion that invasion of the blood stream in septic infections means a fatal issue.

Recent perfections in the technic of blood-culturing have demonstrated beyond reasonable doubt that most specific infections are in reality bacteræmias.

A study of the results of blood cultures is interesting. The earlier observations in typhoid fever, scarlet fever, and streptococcic infections, in rheumatism, endocarditis, pneumonia, and other septic conditions show a low percentage of positive findings, while the more recent works show a very much higher percentage of positive results. In nearly every instance the difference in the results is due to improved methods. These include the use of larger quantities of blood for inoculation, and more particularly the use of more favorable culture media.

In scarlet fever streptococcæmia has been demonstrated during life by Hektoen, Jochmann, and others.

Baginsky and Sommerfeld demonstrated streptococci in the blood of every one of eighty-two cases of scarlet fever examined post mortem.

Bertelsmann found numerous bacteria in the blood during urethral fever which followed the passage of sounds in cases of stricture. In most instances they rapidly disappeared, but in two cases the bacteræmia persisted.

In a case of acute follicular tonsilitis Rosenow isolated the streptococcus pyogenes from the blood during the initial chill, but not subsequently. In two cases of empyema which recovered, cultures from the pus and blood yielded streptococci pyogenes of high virulence in one case and pneumococci in the other.

Five cases of puerperal sepsis out of eight examined contained streptococci in the blood. Two of the cases with the streptococcæmia and one in which the blood cultures were negative died; the rest made uneventful recoveries.

In lobar pneumonia, pneumococci have been demonstrated in the blood in a large percentage of cases by Badnel, Prochaska, Fränkel, Rosenow, and others.[1]

Positive blood cultures have been obtained in a small percentage of cases by Cole, Libman, Kohn, and Sello. Rosenow, Badnel, and Fränkel attribute very little prognostic significance to the mere demonstration of pneumococci in the blood, finding them alike in the fatal and non-fatal cases, while Cole, Libman, Kohn, and Sello regard a pneumococcæmia as a bad prognostic sign, because of their higher percentage of positive findings in the fatal cases.

In this connection it should be stated that blood cultures in cases of post-operative pneumonia are, as a rule, negative unless the pneumonia is of the frank, outspoken, lobar type, when the pneumococcus is usually obtained in pure cultures.

In typhoid fever the bacillus typhosus has been cultivated from the

[1] The total number of cases Rosenow has examined thus far is 300 and the high percentage of positive findings above reported is maintained.

blood in about eighty per cent of cases by Cole, Schotmüller, Auerbach, and others.

The highest percentages of positive cultures are obtained during the first week of the infection, often before the appearance of the agglutination reaction, thus making it a most valuable diagnostic procedure in the differentiation of septic conditions which resemble typhoid fever.

In paratyphoid fever similar results have been obtained. Among other conditions in which a blood culture is often the means of making a correct diagnosis should be mentioned malignant endocarditis, gonorrheal sepsis, cerebro-spinal fever, and other septic states of obscure character.

A positive blood culture in any infection is final from a diagnostic viewpoint. On the other hand, a negative result is of doubtful value and does not prove the absence of micro-organisms in the blood. By a simple modification of the technic, Fränkel and Kinsey (*Jour. Am. Med. Assn.*, 1904, 759) changed their percentage of positive findings in pneumonia from twenty to eighty in the same epidemic. Similar results have been obtained in typhoid fever.

Anyone with an understanding of bacteriological principles can make a blood culture. It is a perfectly harmless procedure when properly carried out. There is no danger of thrombosis. The following steps should be followed:

1. **Preparation of the Arm.**—Constrict the arm by means of an elastic rubber tube to the extent of producing venous stasis, care being exercised not to interfere with the arterial circulation. This is likely to happen in severe septic conditions when the pulse is of small volume and of low tension. Locate the median basilic or median cephalic vein. The point selected for the puncture should be near the median line, so as to avoid the external and internal cutaneous nerves. The former lies just beneath the outer end of the median cephalic vein, while the latter crosses the median basilic at its inner end.

The skin should be sterilized with ninety-five per cent alcohol. The superficial epithelium should be rubbed off. If this is done thoroughly, there need be no fear of infecting the patient or of contaminating the culture media. The more elaborate methods for sterilization are unnecessary, and as they are time-consuming they are undesirable, especially for routine work.

2. **The Puncture and Withdrawal of the Blood.**—The venous puncture is usually easy, provided a needle with a sharp point is used. Those fitting a glass syringe of the Luer type, having a capacity of not less than 10 c.c., are very satisfactory. For sterilizing the syringe and needle the autoclave is to be preferred. The syringe and needles should be boiled for at least one half hour in order to prevent con-

tamination. After the syringe is filled the constrictor should be removed before withdrawing the needle. Moderate pressure should be made over the site of the puncture with a dry sterile sponge until the blood clots. This may be done by the patient while the operator inoculates the media.

The blood clot which forms is sufficient protection for the small, punctured wound. A small collodion dressing may be applied, but it is unnecessary. The minute blood clot exposed to the air protects the vein more securely against bacterial infection than when covered with collodion.

3. **The Inoculation of the Media.**—The strictest precautions should be observed, for it is during this procedure that contamination is most apt to occur. The needle which has been passed through the skin is apt to carry with it a smaller or larger number of staphylococci, no matter what method of sterilization is used. It should therefore be removed and inoculation made through the sterile glass end of the syringe. The tubes or flasks containing the media should be held as nearly horizontal as possible during inoculation. The mouths should be flamed thoroughly and the cotton plugs replaced as soon as possible.

The character of the media is of the greatest importance. Litmus milk and beef broth are the most favorable for routine work. The reaction of the latter should be one per cent acid to phenolphthalein or neutral to litmus.

The broth should be made from meat and not from the extract of beef. It should be sterilized by fractional sterilization instead of by the autoclave. This is particularly important when a pneumococcus infection is suspected.

Dilution of the blood, while of lesser importance, should be taken note of. A convenient way to control this factor in routine work is to take four flasks, each containing 50 c.c. of the media, and add approximately one, two, three, and four or more c.c. of blood to each flask, respectively. The inoculated media are then placed in the thermostat. At the end of twenty-four hours the cultures usually show the presence of a growth if the result is to be positive. Very exceptionally, positive results are obtained first after three or four days have elapsed.

EXAMINATION OF BLOOD FOR HÆMATOZOA

Attention to the microscopic examination of the blood for animal parasites in puzzling septic conditions will frequently lead to a correct diagnosis.

Pernicious malaria without definite paroxysms and with an atypical course often resembles typhoid fever, meningitis, uræmic coma, perni-

cious anæmia, tuberculosis, and dysentery. An examination of the blood will usually reveal the malarial organism.

In tropical countries the spirillum of Obermeier, filaria sanguinis hominis and trypanosoma Gambiensi should be searched for. All of these occur in the peripheral blood, while the Leishman-Donovan bodies of tropical splenomegaly are usually obtained by splenic puncture, since they appear only exceptionally in the general circulation.

LEUCOCYTOSIS

The term leucocytosis has come to mean the presence in the blood of an increased number of white blood corpuscles of the same variety morphologically as those in normal blood. Usually the greatest increase is in the polymorphonuclear neutrophiles, and this is sometimes spoken of as a polymorphonuclear leucocytosis.

A distinct diminution of leucocytes is often spoken of as a leucopenia or hypoleucocytosis in contrast to hyperleucocytosis, indicating an excess of cells.

Much experimental and clinical work has been done in recent years upon the significance and value of inflammatory and infectious leucocytosis.

The work of Metschnikoff and his school upon phagocytosis and allied subjects has taught us to look upon a leucocytosis in many infections not only as an expression of the intensity of the irritant, but as a positive means of defense. The investigations of Wright and Douglas, Hektoen, Ruediger, Rosenow, Potter, Dittman, Bradley, and others have thrown much light upon the mechanism involved. They have shown that destruction of various bacteria in the test tube is a result of the combined action of the serum, " opsonin," and the living cell, the leucocyte. They have pointed out that opsonification, phagocytosis, and intraphagocytic digestion probably play an important rôle in combating certain infections.

If a leucocytosis were constantly present in the same disease and always absent in certain others, and if the maxim that " the higher the leucocytosis the more favorable the prognosis " were always true, there would be no occasion for the diverse opinions held by different observers as to the value of leucocytosis as a prognostic sign. But since a high leucocytosis may be an expression of a severe infection and at the same time be an index of resistance, and because in overwhelming infections it often fails to appear from the beginning or later disappears, there is ample reason why authors differ as to its value.

When we remember that a pathological leucocytosis may be inflammatory or infectious, post-hæmorrhagic or toxic in nature or the result

of malignant disease, it is obvious that enumeration of the leucocytes can help us in the diagnosis and prognosis of disease generally, and especially in the diagnosis and prognosis of many surgical conditions, only when correlated with the other clinical data. When this is done, leucocytosis is often of the greatest value and furnishes the necessary missing link in a chain of evidence requisite for the correct diagnosis of some obscure internal infection.

The degree of leucocytosis varies greatly from day to day in many cases of sepsis. A single leucocyte count is of very little more value than a single temperature or pulse record. Leucocytosis should be studied from day to day or oftener in curves much in the same way as the temperature and pulse are.

If this is done and the observations are correlated with the clinical symptoms, the leucocyte curve will not infrequently furnish the data necessary to decide when to institute surgical interference in a given case and when not to do so.

Before rendering a final decision as to the significance of a leucocytosis the various forms of purely physiological leucocytosis must be ruled out. A digestion leucocytosis is usually at its maximum about four hours after a meal. The increase rarely exceeds 3,500 cells. It does not occur when leucocytosis is already present.

Leucocytosis is of very little value in the differential diagnosis of ulcer and carcinoma of the stomach, because it fails to appear in ulcer quite as commonly as in beginning carcinoma.

The leucocytosis of the newborn, of pregnancy, and of parturition, as well as that following certain mechanical and thermal influences, must also be borne in mind.

Busse, King, and others have shown that a post-operative leucocytosis of from 5,000 to 10,000 leucocytes per cubic millimeter in from six to thirty-six or forty-eight hours is normal provided it is not maintained. A persistent leucocytosis in aseptic operations must be looked upon as indicating infection, defective drainage, spreading inflammation, or hæmorrhage.

The significance of lymphocytosis, eosinophilia, myelæmia, iodophilia, and other cellular changes of the blood are omitted here because of their relatively limited value to the surgeon.

COAGULATION OF THE BLOOD

The time required for the coagulation of the blood after withdrawal from the body is dependent upon a number of conditions. The coagulation is slower when the blood is obtained from a deep cut or from venous puncture than when it flows from a superficial cut. Coagula-

tion is dependent upon the amount of blood allowed to flow, upon the pressure used, upon the temperature, and, to a considerable degree, upon the nature of the containing receptacle.

The time of day makes a difference, the coagulation time being perceptibly shorter in the morning than in the afternoon. The test should not be made shortly after a meal, since the time of coagulation is influenced by certain foods and drugs. Uniformity of technic and the necessity of always stating the method employed in the report of cases cannot be too strongly emphasized.

Too much reliance should not be placed upon the coagulation time. Only relatively great deviations from the normal should be considered, for it has been proven that coagulation outside of the body is not the same process as intravascular coagulation. Welch justly says that " we cannot bring the appearance of coagulation in the living vessel into direct parallel with coagulation of the blood as ordinarily understood."

Extravascular coagulation unquestionably is dependent to some degree upon the fibrin content, while intravascular coagulation is quite independent of it. This is indicated by the fact that in cachexia, anæmia, and typhoid fever thrombosis is common, yet the fibrin content of the blood is low, while in pneumonia and acute articular rheumatism, where the fibrin content is high, thrombosis is rare.

The time required for a clot to form in wounds (the point of greatest interest to surgeons) corresponds, however, relatively closely to the time of coagulation as determined by laboratory methods; hence their value.[1]

The coagulation time is prolonged in many cases of jaundice, anæmia, anasarca, hæmoglobinæmia, hæmophilia, purpura, asphyxia, acute alcoholism, cobra poisoning, and in some toxic conditions.

It is interesting to note here that the lengthened coagulation time in jaundice appears to depend not upon the jaundice *per se*, but rather upon the associated toxæmia, hæmoglobinæmia, and excessive anæmia. Fatal post-operative hæmorrhage is more prone to occur in cases of malignant disease of the biliary passages with jaundice than in the obstructive jaundice associated with cholelithiasis.

Coagulation is hastened by the administration of relatively small doses of the calcium salts; retarded when these salts are given in too large doses or for too long a time. From 60 to 90 grains daily of calcium chlorid for three or four days will usually promote clotting in a patient whose coagulation is delayed.

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[1] For the various methods used the reader is referred to works on hæmatology and laboratory diagnosis.

In cases of long-standing jaundice with delayed coagulation the coagulation time should, when possible, be brought within five minutes before an operation is performed.

Early in typhoid fever, delayed coagulation may predispose to intestinal hæmorrhage, while in the later stages of this infection the coagulability of the blood may be so increased as to favor thrombosis. This tendency to rapid coagulation is believed to be due to the excessive quantity of calcium salts in the blood of the convalescent typhoid, the result of the prolonged milk diet. Wright and Knapp suggest, in order to prevent thrombosis in this disease, the partial decalcification of the milk by the addition of sodium citrate as soon as the danger of hæmorrhage is over.

HÆMOGLOBIN AND ERYTHROCYTES

A relatively greater diminution in the hæmoglobin than in the red cells occurs in the symptomatic anæmias attending the chronic constitutional diseases, in chlorosis, infections, hæmorrhagic disorders, and the various toxic states.

Because the hæmoglobin reduction in these conditions is greater than that of the red cells, the amount of hæmoglobin per red corpuscle is less than normal. This condition is known as a low color index. The color index is obtained by dividing the percentage of hæmoglobin by the percentage of red cells present. It is important in all cases of anæmia to determine this point, since the graver anæmias and leukæmias have a normal or even a high color index; the low hæmoglobin reading in these cases being due to the reduction in the number of red cells.

Pallor of the skin is not necessarily due to anæmia. It may be due to a deficient cutaneous circulation, the result of valvular disease, myocarditis, or vasomotor disturbances, and hence should never be used as an index of the hæmoglobin content of the blood.

In the interpretation of hæmoglobin values it must be remembered that concentration of the blood may account for abnormally high figures, while in dilute hydræmic blood the reverse occurs, the gain or loss in either instance paralleling the fluctuations of the erythrocytes.

Individuals with a low hæmoglobin reading do not bear general anæsthesia and the loss of blood incident to an operation well. Bierfreund, Mikulicz, and others believe that a hæmoglobin percentage below thirty or forty contraindicates a general anæsthetic. Numerous reports of successful operations under general anæsthesia in cases in which the hæmoglobin percentages ranged between fifteen and thirty have been made, but all agree that operations should be performed as a life-saving

measure only when the hæmoglobin is so low. Nowhere in the realm of surgery does the skill of the operator and anæsthetist count for so much.

Bergmann, Bauman, Aborti, and others have shown that iron is the most useful blood builder for patients deficient in hæmoglobin, hence it should be given freely in secondary anæmias. Arsenic stimulates indirectly the hæmogenic centers, and is therefore of greater value when the deficiency in hæmoglobin is the result of a diminution in the number of erythrocytes. It has little or no effect in exciting directly a hæmoglobin increase.

CRYOSCOPY

The freezing point (expressed by the Greek delta, Δ) of normal blood ranges between −0.56° and −0.58° C., while normal urine freezes between −0.9° and −0.2° C. These fluids are no exception to the law that the greater the molecular concentration of liquids the lower the freezing point.

Surgically, cryoscopy is used chiefly in determining the integrity of the kidneys.

Koranyi showed that in diseases of the kidney with renal insufficiency the Δ of the blood falls, while that of the urine correspondingly rises, the blood becoming surcharged with excrementitious matter, which the crippled kidneys fail to discharge.

Kümmel, Lindeman, and others assert that a freezing point below −0.58° or certainly −0.6° is a distinct contraindication to a nephrectomy, because they believe that when this figure is obtained both kidneys are too extensively implicated to insure adequate elimination when one kidney is removed. This view has been revised because Tieken, Loeb and Adrian, Rovsing, and others have shown that unilateral lesions may cause decided abnormalities of the Δ, while bilateral lesions may exist without any such change. Their studies have shown that cryoscopy alone is of doubtful value in determining the state of renal activity, because other factors modify the molecular concentration of the blood and urine, such as circulatory stasis, dependent upon cardiovascular, hepatic diseases, abdominal neoplasms, and anæmia from any cause. A lowering of the Δ of the blood, while not accepted universally as a contraindication to nephrectomy, should always make the surgeon cautious. This question, as well as other clinical features of cryoscopy, has been extensively studied by Tieken, Ogsten, Casper and Richter, and Rinker.

LITERATURE.—*Badnel.* Rev. de Méd., 1899, p. 70.—*Baginsky* and *Sommerfeld.* Arch. f. Kinderheilkunde, 1902.—*Cole.* Bull. Johns Hopkins Hosp., 1901, XII, 203; 1903, XIII, 136.—*Hektoen.* Jour. A. M. A., 1903, XL, 685; Jour. Infectious Dis., 1906,

III, 156.—*Jochmann.* Zeitschrift f. klin. Med., 1905, LV, 316.—*Kinsey.* Jour. A. M. A., 1904, 759.—*Kohn.* Deutsch. med. Wochenschrift, 1897, XXIII, 186.—*Libman.* Jour. Med. Research, 1901, I, 84.—*Prochaska.* Centralblatt f. inner. Med., 1900, XXI, 1145.—*Rosenow.* Amer. Jour. of Obstetrics, 1904, 762; Jour. Infectious Diseases, 1904, 280; 1906, III, 683.—*Schotmüller.* Deutsch. med. Wochenschrift, 1900, Aug. 9.—*Sello.* Ztschrft. f. klin. Med., 1898, XXXVI, 112.—*Wright* and *Douglas.* Proceedings of Roy. Soc., 1903, LXXII, 357; 1904, LXXXIII, 128.

III. WOUND INFECTIONS OF DIFFERENT ORIGINS AND SURGICAL INFECTIOUS DISEASES

CHAPTER I

WOUND INFECTIONS CAUSED BY POISONS

Poisoning by Insects, Snakes, etc.—Intoxications, varying in severity, may follow the sting or bite of a number of different insects (bee, wasp, hornet, spider, gnat, flea, bedbug, and others). Besides the local inflammatory reaction which follows the sting or bite, there may be general symptoms, such as superficial respirations, rapid pulse, faintness, collapse, and vomiting. These general symptoms are most apt to develop when a man or animal has been attacked by a swarm of bees or wasps, and the lesions are distributed over a large part of the surface of the body. Usually the general and local symptoms subside rapidly, but the patient may feel weak and feeble for several days. Death has followed, however, a single sting by a bee or wasp.

The sting, which is situated in the posterior end of the bodies of bees and wasps, together with the poison bladder, is frequently left in the wound, and should always be removed. The wound should be touched with a dilute solution of ammonia in order to neutralize the poison, which contains an acid (probably formic acid). The same method should be employed in the treatment of stings by the European scorpion, which are very similar to stings inflicted by bees and wasps. The application of naphthalene has been recommended in the treatment of mosquito bites (Voges).

[" Poisonous snakes are widely distributed in all countries of the temperate, and especially of the torrid, zones. In the United States about seventeen species of rattlesnakes and ten species of copperheads and moccasins, vipers, coral, and harlequin snakes, etc., are classed as poisonous; with them a Texan reptile known as the Gila monster is also classed. In South America, Central America, Africa, the West Indies, and Australia many venomous reptiles are found. In Europe the adder and viper are dreaded, while in India much attention has been paid to the Thanatophidia, the cobra having furnished the venom upon which

the work of Fraser, Calmette, and others has been based."—Keen's "Surgery," Vol. I, pp. 539 and 540.]

["The poison apparatus of snakes consists of a secretory gland on each side which communicates with a tubular fang by means of a duct. In the passive state the fangs are directed backward on the roof of the mouth, but when the animal strikes, their points are made to project forward and the poison is forced through the canals by muscular compression of the sac. The venom is a glandular secretion."—Ricketts, "Infection, Immunity, and Serum Therapy," pp. 264 and 265.]

Bites caused by poisonous snakes may be recognized by two small punctured wounds lying side by side, while a zigzag wound is produced by non-poisonous snakes.

Action of Snake Venom.—Snake venom, like the toxins produced by bacteria, dissolves red blood corpuscles, and contains two toxic albuminous bodies (toxalbumins) which produce a local and general reaction.

["The venoms of different snakes vary a great deal in their toxic properties. The most important constituents are those which attack the nervous system (neurotoxin), the blood corpuscles (hæmolysins and hæmagglutinins), and the endothelium of blood vessels causing hæmorrhages (hæmorrhagin, an endotheliotoxin). The neurotoxin causes death by paralysis of the cardiac and respiratory centers. The hæmolysin appears to be of less importance as a cause of death.

"The venoms of the cobra, water moccasin, daboia, and some poisonous sea snakes are essentially neurotoxic, although they have strong dissolving powers for the erythrocytes of some animals. In studying the hæmolytic powers of the venoms of cobra, copperhead, and rattlesnake, Flexner and Noguchi found cobra venom to be the most hæmolytic, and that of the rattlesnake the least. They attribute the toxicity of rattlesnake poison chiefly to the action of hæmorrhagin. The same authors studied the action of different venoms on the cells of various animals, and by absorption experiments found independent cytotoxins for the testis, liver, kidney, and blood. Not only was there a distinct cytotoxin for each organ of an animal, but also for the same organ of different animals, results which speak for a remarkable complexity of venom. Certain venoms contain a leucocytic toxin.

Proteolytic Ferments.—"That venoms contain proteolytic ferments is shown by their ability to digest gelatin and fibrin. This power may be related to the softening of the muscles which has been noted clinically in cases of poisoning. The rapid decomposition of the body which follows death by snake-poisoning is associated with a decrease in the bactericidal power of the blood, which, according to Flexner and No-

guchi, depends on fixation of the complement by the venom.''—Ricketts, '' Infection, Immunity, and Serum Therapy,'' pp. 265 and 266.]

Symptoms: Local and General.—The local symptoms consist of painful swelling of the tissues surrounding the wound, which develops soon after the bite is received. The skin covering the swollen tissues is not discolored at first, but petechiæ and suggillations soon develop. In a short time the swelling becomes very extensive, and within half an hour the extremity becomes twice its normal size. The lymphangitis and lymphadenitis which frequently develop are due to the absorption of the venom; suppurative phlegmonous inflammation to secondary infection with pyogenic bacteria. Necrosis and gangrene of the œdematous tissue are frequently produced by a too long-continued and too great constriction of the extremity or by subsequent putrefactive infections. In a few hours after the injury the general symptoms, consisting of dizziness, faintness, fever, headache, small rapid pulse, dyspnœa, the feeling of anxiety, vomiting, diarrhœa, with or without blood, and collapse, develop. In the majority of cases these symptoms disappear in a few days (on an average of nine days, according to W. K. Müller) and the patient recovers, although a marked weakness may persist for a long time. Death due to cardiac weakness or asphyxia occurs in from three to nine per cent of the cases. The coagulability of the blood is reduced in these fatal cases, and numerous hæmorrhages are found in the viscera and intestinal mucous membranes.

Comparative Toxicity of Venoms.—The local and general symptoms following bites inflicted by the rattlesnake (America) and the cobra (Asia, Africa) are much more severe than those following bites inflicted by the viper, and end fatally more frequently. The mortality following bites inflicted by these two snakes is about twenty per cent. In India more than 20,000 people die each year as the result of snake bites. Immediate death may follow injury of a vein, with direct injection of the venom into the circulation. The cases of ordinary severity end fatally in a few days, the patient becoming delirious and unconscious, and tetanic convulsions developing. In chronic cases death may follow the after-effects of the venom (cachexia, tendency to œdema, hæmorrhagic diathesis) after months or years. It is important to know that the venom in museum specimens never becomes inactive.

Treatment: Local and General.—The laity have recognized for a long time that the symptoms following snake bites are due to absorption of venom, and have formulated two important rules to prevent or lessen the absorption of the poisonous material: (1) To immediately suck the wound, removing the venom, or to express it by digital pressure applied to the tissues about the wound; (2) to tie off the injured part (for example, an extremity) close to the bite as soon as possible, and in this

way prevent the absorption of the venom until some other treatment can be instituted. It is dangerous to suck a wound unless one has a special apparatus of some kind or can protect the lips, as fissures may become infected. Venom has no effect upon healthy mucous membranes, and even if introduced into the stomach is rendered harmless.

The layman has outlined the work for the physician, whose duty it is to excise the wound as soon as possible and to make large incisions into the inflamed, and, if a constrictor has been applied, œdematous tissues, providing in this way an escape for at least a part of the venom. The constricting strap or rope which the patient himself or a friend may have applied should not be removed until incisions have been made and considerable serum has exuded, for in this way the absorption of considerable amounts of venom which might prove dangerous is prevented. Incised wounds secrete more profusely than those covered with a crust or membrane; therefore a knife is preferred to caustics and the actual cautery in treating wounds of this character. Treatment by incisions is more reliable than the methods intended to neutralize the toxic properties of the venom by subcutaneous injections of chemical agents, such as a one half per cent solution of potassium permanganate or a freshly filtered solution of chlorinated lime. Cupping and scarification may be combined with incisions. Amputation of the smaller parts is sometimes indicated. Of course the incised wounds should be dressed aseptically, immobilizing dressings applied, and the part elevated if possible.

If there is cardiac weakness, stimulants should be administered. Subcutaneous injections of camphor and transfusions of physiological salt solution (0.9 per cent) act favorably. Large doses of alcohol (punch, mulled wine, cognac, whisky) are in great repute among the laity.

Antitoxic Sera.—Calmette was the first to attempt to produce an antitoxic serum for the treatment of snake bites. [" Calmette's antivenin is obtained by immunizing horses with a mixture of venoms (eighty per cent cobra, twenty per cent viperine venom) which are attenuated before injection. Six months are required to produce a strong antivenin. The claim of Calmette that his serum is effective against all snake venom is erroneous. It neutralizes those venoms the toxicity of which depends largely on neurotoxins and hæmolysins, but has little influence on rattlesnake poison, the essential toxin of which is hæmorrhagin. Antivenin for rattlesnake and water moccasin may be prepared by immunizing with the corresponding venoms which have been attenuated by weak acids. Noguchi has produced serum of such strength that it promises to be of practical value in the treatment of rattlesnake bites.

"As indicated previously, the action of venom is preceded by no appreciable incubation period; hence an antitoxin to be effective must be administered not later than a few hours after the bite has occurred. Noguchi found in relation to antivenin for the rattlesnake that the amount necessary to save experimental animals was quadrupled three hours after intravenous injection of two fatal doses of venom. Fortunately the venom is less toxic when introduced subcutaneously."—Ricketts, "Infection, Immunity, and Serum Therapy," pp. 267 and 268.] The bile of poisonous snakes has an antitoxic action (Fraser); likewise the serum of animals immunized against tetanus and hydrophobia (Roux).

Indian Arrow Poison.—Wound infections may be produced by the poisoned points of arrows used by savages. Vegetable (strychnin, antiarin in Asia, strophanthus in Africa) or animal poisons (rattlesnake venom mixed with decomposing meat or blood in America) are most frequently employed for this purpose. These poisons paralyze the heart or cause tetanic convulsions. As a rule, death follows their action in a short time. If the poison is sucked out of the wound immediately the life of the individual may be saved. It is important to use stimulants when the poison acts upon the heart. Curare, the arrow poison most highly valued by the Indians, paralyzes all the voluntary muscles.

Cadaveric Poisoning.—The wound infections caused by cadaveric poisons are purely toxic in character, and are much rarer than was formerly considered to be the case. Although cadaverin, which belongs to the ptomains, has, according to Grawitz, a pyogenic action, the acute, severe infections which follow injuries received during post-mortem examination of fresh cadavers are caused by highly virulent bacteria. The latter are rapidly absorbed and produce more frequently acute, severe, general infections than local suppurating lesions. Most of these severe infections follow injuries received during post-mortem examinations of subjects dying of virulent bacterial infections (peritonitis, meningitis, general infections). The wounds received during the dissection of and operations upon old cadavers are rarely followed even by local inflammation if the hæmorrhage from the wound following compression of the surrounding tissue is free and a dressing is applied, which prevents secondary infection. Billroth has recommended concentrated acetic acid as the best caustic for this class of wounds.

LITERATURE.—*Brenning.* Die Vergiftungen durch Schlangen. Enke, Stuttgart, 1895.—*Brieger.* Ueber Pfeilgifte aus Deutsch-Ostafrika. Berl. klin. Wochenschr., 1902, p. 277.—*Calmette.* Comptes rendus, 1896, Nr. 4; Münch. med. Wochenschr., 1896, p. 936.—*Fraser.* Die antitoxischen Eigenschaften der Galle von Schlangen. Wiener med. Blätter, 1897, p. 481.—*Grawitz.* Ueber die Bedeutung des Kadaverins

für die Entstehung der Eiterung. Virchow's Arch., Bd. 110, 1887, p. 1.—*Husemann.* Behandlung der Vergiftungen. Handbuch der spez. Therapie innerer Krankheiten von Penzoldt und Stintzung, 1895, Bd. 2.—*Lamb.* Die Serumbehandlung der Schlangenbisse. Lancet, Nov. 5, 1904.—*Lewin.* Die Pfeilgifte. Histor. u. experim. Untersuchungen. Reimer, Berlin, 1903.—*W. K. Müller.* Die Verletzungen durch Schlangenbiss in Pommern. I.-D., Greifswald, 1895.

CHAPTER II

HYDROPHOBIA (LYSSA, RABIES)

HYDROPHOBIA as it occurs in man is an acute wound infection which invariably proves fatal. It is transmitted to man by the bite of a rabid animal, most frequently (ninety per cent) by the dog, or in its saliva, which in some way is introduced into fresh wounds.

Virus of Hydrophobia: Negri Bodies.—Nothing definite is known concerning the virus of hydrophobia. Negri in 1903 first described round bodies 4 to 10 μ in size in the nervous system of animals dying of the disease. These bodies are found within the large ganglion cells, and are especially numerous in the horn of Ammon and in the cells of the cerebellum. They are also found, but not in as large numbers, in the cells of the medulla oblongata, the spinal cord, and spinal ganglia. Negri's findings have been confirmed by Volperino, Bertarelli, Schiffmann, Maresch, and others. These bodies may be regarded as the most characteristic findings in animals and men dying of hydrophobia. Further investigation must decide whether Negri is correct in regarding them as protozoa and as the specific cause of the disease.

Distribution of the Virus.—It has been demonstrated by animal experiments that the brain, spinal cord, and peripheral nerves contain the virus. It has even been demonstrated in the saliva of animals before they have shown symptoms of the disease. The virus apparently passes from the wound along injured nerve trunks (Babes, di Vestea-Zagari, Wyssokowitch, and others). ["Experimental work shows conclusively that the virus is conveyed to the central nervous system by means of the peripheral nerves, and that the infection is closely associated with the wounding of nerves. It has been shown that if wounding of nerves is entirely avoided, as in intraperitoneal injections into rabbits (Marx) the full virulent nervous tissue may be used for immunization." —Ricketts, "Infection, Immunity, and Serum Therapy," p. 517.] The infection develops most rapidly and most frequently after the injection of small amounts of the brain or spinal cord of patients or animals dying of the disease into the subdural space. Infection does not follow

subcutaneous injections, and the results of intravascular injections are inconstant. This partly demonstrates that the virus is taken up by and extends along injured nerves.

Susceptibility of Different Animals.—All warm-blooded animals are susceptible. The disease may be transmitted to man by the dog, wolf, cat, and fox. Direct infection from man to man is not known.

Hydrophobia in Dogs.—In dogs the incubation varies from three to five weeks. The prodromal stage is characterized by restlessness, loss of appetite, nausea, and irritability. Then in a few days the symptoms characteristic of the second stage of the disease become pronounced. [" According to Bollinger the initial or prodromal stage lasts from one half to two or three days, and the stage of real madness, irritation, or maniacal stage lasts three to four days."—Tillman's " Text-book of Surgery," I, 398.] The virus may be transmitted to man and animals through the saliva before there are any symptoms of the disease. The bite of an animal which is apparently healthy may therefore carry with it the danger of infection.

Two forms of hydrophobia may be distinguished in dogs—*the raging* and the *paralytic*. According to Pasteur, the raging form develops when the virus attacks chiefly the brain, and the paralytic form when it attacks chiefly the spinal cord. In the convulsive or maniacal form the disposition of the dog changes suddenly. The animal becomes more irritable, attempts to bite other animals or surrounding objects, runs confusedly about, and utters long-drawn-out howls, emaciates rapidly, and shows a preference for indigestible things, such as wood, earth, and fæces. A pharyngeal spasm develops at every attempt to drink, therefore the name hydrophobia, meaning " fear of water," has been given the disease. The third stage (stage of paralysis) develops upon the third or fourth day. The hind legs first become paralyzed. The paralysis later extends to other muscles, and on from the third to the sixth day convulsions develop and death occurs. An animal which develops hydrophobia never recovers. The paralytic form is still more rapid; the paralysis (particularly of the muscles of the extremities, mastication, and deglutition) develops earlier, as there is no stage of excitement.

Hydrophobia in Man.—Hydrophobia in man is characterized by a long period of incubation, generally from twenty to sixty days. It varies from fifteen days to six months. A longer period of incubation than six months is extremely rare. Hydrophobia does not follow every bite by a rabid dog, as the clothes afford some protection against the infectious saliva. Only a small proportion of those bitten by rabid animals (according to Babes, not one third; according to others, still less) develop the disease. Hydrophobia follows less frequently bites of protected parts of the body, more often bites of the head and face.

Clinical Course.—The prodromal stage begins with pain in the wound or scar, which radiates along the nerves supplying the surrounding area. Sometimes the scar becomes reddened, sometimes when the wound is not healed the granulations are unhealthy. Loss of appetite, headache, melancholia, restlessness, anxiety, sleeplessness, slight dysphagia and dyspnœa, sometimes early aversion to liquids, in spite of great thirst, and slight temperature indicate the beginning of the disease. After a few hours or days, painful pharyngeal spasms develop at every attempt at drinking and eating. These pharyngeal spasms, which finally may be even provoked by the sight of a drinking glass, render the swallowing of food, even the swallowing of saliva impossible, and are the principal symptoms of the hydrophobic stage which is characterized by an increased reflex excitability. At this time the skin and sense organs are hypersensitive, and any irritation, such as stroking or blowing upon the skin, loud noises, strong light, etc., produces a dyspnœic condition and clonic spasms of all the muscles. The pupils become dilated when the skin is irritated or the auditory nerve is stimulated (Schaffer). When this increased excitability extends from the spinal cord and medulla to the brain, the reflexes are abolished, the dilated pupil does not react, the urine is discharged involuntarily, and delirium develops. Sometimes extensive paralysis occurs (lumbar and cervical paraplegias) and sometimes the last or convulsive stage may begin with violent and persistent vomiting and be characterized by convulsions. The patient rapidly becomes exhausted, a high fever develops, and death occurs, most frequently between the second and fourth days of the disease. Consciousness may be retained until the end.

Pathological Anatomy.—Inflammatory and degenerative changes have been described in the motor centers of the central nervous system, especially in the spinal cord (acute myelitis). These changes are most advanced in the segments which correspond to the nerves primarily involved (Schaffer). [Babes has described peculiar perivascular nodules in the medulla and spinal cord composed of lymphoid cells; van Gehuchten, a proliferation of the endothelium surrounding the ganglion cells. Degenerative and atrophic changes occur in the latter.]

Diagnosis.—Hydrophobia may be mistaken for head tetanus, as pharyngeal spasms occur in the latter also. Hysteria must be considered in making a diagnosis.

Treatment.—In the treatment of bites of rabid or supposedly rabid animals an attempt should be made to render the virus harmless as soon as possible. Excision of the wound, amputation of small parts, and open treatment of the wound are most efficacious. Cauterization of the wound is not safe, as the eschar prevents the discharge of wound secretion, and the retained virus develops beneath it. The same thing hap-

pens when the primary wound or the one following its excision is sutured. The edges of large and deep wounds, the tissues of which are contused and lacerated, should be trimmed off and a tampon which provides for the discharge of the secretion should be inserted.

Because of the longer incubation period local treatment is of more value in man than in animals. Babes found that in order to prevent the development of the disease in animals, the wound must be cauterized with a Paquelin cautery not later than five minutes after infection.

If the disease has developed, narcotics should be given to control the painful spasms. Hourly subcutaneous injections of curare (one fifth to one half grain) have been recommended (Penzoldt). Rectal and subcutaneous injections of physiological salt solution should be given to control the thirst.

The disease has almost disappeared from Germany and England. The police regulations against stray and suspected animals are very rigid, and there are laws which provide for the muzzling of dogs. According to M. Kirschner there were, on an average, only four deaths a year from hydrophobia in Prussia in the period between 1889 to 1899. In England there have been no deaths for several years. In America hydrophobia is still common. In Chicago from fifteen to twenty deaths occur each year.

The long incubation period in man is taken advantage of in the Pasteur treatment, as an immunity may be established against the virus before the symptoms develop and the disease may be prevented. Pasteur gave the name of street virus (*virus de rue*) to that obtained from the nervous system of dogs in which the disease develops spontaneously. When the street virus is injected subdurally into rabbits, they develop the disease after two or three weeks. When this virus is passed through a number of rabbits the incubation period is reduced finally to six days. It is impossible to reduce the incubation period below six days, and the virus obtained from the nervous tissue of such an animal is extremely virulent. It is called the fixed virus (*virus fixé*). In the Pasteur method this virus is gradually attenuated by drying the spinal cord. The virus obtained after drying the cord for fourteen days is the weakest and is no longer active for rabbits. Dogs, which are more susceptible than man, may be rendered immune against highly virulent virus, if bouillon emulsions of a fourteen day old cord, then a thirteen, twelve, and so on are injected daily; in other words, if the virulence of the virus injected is gradually increased each day.

The Pasteur treatment is the only one, according to our present knowledge, which will prevent the development of the disease. It cannot be relied upon when the incubation period is short (about two weeks), as is frequently the case in bites of the face and in those in-

flicted by wolves, or when treatment is instituted late, so that only two or three weeks elapse before the symptoms develop. However, if the injections are given in rapid succession, an immunity against the strongest virus may be established as early as the third day, and cures have repeatedly been made. Ten c.c. of the emulsion should be injected subcutaneously each time in the hypochondrium. After the protective inoculations have been completed the blood contains protective substances (Kraus and Kreisl).

Mortality.—The mortality has been considerably reduced since people bitten by rabid or supposedly rabid dogs have been subjected to the Pasteur treatment. Pottevin estimates the mortality among 13,817 patients treated in Paris as 0.5 per cent, while the mortality among patients not treated is not lower than 10 per cent. Högyes gives the mortality of those who received treatment as 0.85 per cent, of those who did not as 14.14 per cent. In the dangerous wolf bites, 90 per cent of which are followed by the disease, the results following early treatment, a mortality of from 10 to 15 per cent are very favorable (Babes).

Injections of blood serum obtained from immunized animals have also been made with success (Babes, Tizzoni, Schwarz).

LITERATURE.—*Babes.* Studien über die Wutkrankheit. Virchow's Arch., Bd. 110, 1887, p. 562;—Ueber die Behandlung von 300 von wütenden Wölfen Gebissenen. Zeitschr. f. Hygiene, Bd. 47, 1904, p. 179;—Behandlung der Wutkrankheit des Menschen. Im Handb. der spez. Therapie von Penzoldt u. Stintzing, 1903, Bd. 1; *Bertarelli.* Die Negrischen Körperchen im Nervensystem der wutkranken Tiere, ihr diagnostischer Wert und ihre Bedeutung. Zentralbl. f. Bakteriol., Bd. 37. Abstract.

CHAPTER III

TETANUS: LOCKJAW

NICOLAIER in 1884 produced fatal tetanus in mice, rabbits, and guinea pigs by infecting them with garden earth, in which he had demonstrated a bacillus with a somewhat rounded end. Rosenbach (1885) found a similar bacillus in the wound of a patient sick with tetanus. Kitasato (1889), using anaërobic culture media, was the first to obtain pure cultures of the bacillus and to produce with the cultures experimental tetanus.

Bacillus of Tetanus.—The tetanus bacillus is a slender, slightly motile organism which develops a terminal spore, and for this reason the bacillus with its spore resembles a drumstick. The bacillus frequently develops filamentous forms in cultures. It stains readily with the ordi-

nary stains and also by Gram's method. The bacillus is widely distributed in the ground, and is found as far as 30 cm. below the surface, being carried probably to this depth in the dung of animals, in which it is frequently found. Apparently the bacilli find conditions favorable for growth in the intestines of animals, but tetanus does not develop from the intestine, as feeding experiments have demonstrated. Of the domestic animals, the horse, cow, and sheep develop the disease most frequently after injury (or after castration).

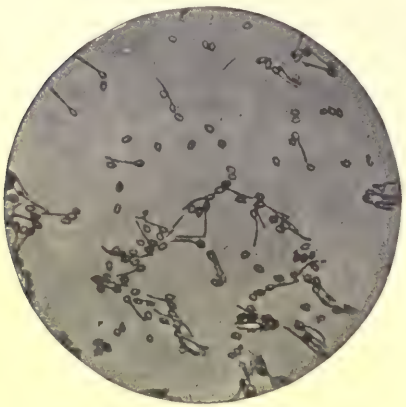

Fig. 134.—Tetanus Bacilli.

The bacilli are obligatory anaërobes, and grow best at 98.5° F. Yellow colonies having irregular offshoots which grow out into the medium appear in gelatin and agar on the second day. Gelatin about stab cultures slowly liquefies and gas is formed. Bouillon is clouded. All cultures have a disgusting odor.

Susceptibility of Different Animals.—Guinea pigs, mice, and rabbits are best suited for experimental purposes. Fatal tetanus is easily produced in these animals by the injection of virulent cultures. Cultures become inactive when heated for five minutes at 149° F., and are rendered toxin free, as the toxins secreted by tetanus bacilli are destroyed by heat. The toxin-free cultures still contain viable spores, but are active only when injected in large amounts. The bacilli must therefore be injected with their toxins to obtain results. If old cultures, rich in toxins, or foreign bodies to which bacilli are attached, are employed, a fatal tetanus develops after an incubation period of from one to three days. As a rule, the bacilli do not extend beyond the wound, and only in rare cases have they been demonstrated in the viscera (von Oettingen and Zumpe).

Tetanus Toxins.—Filtered cultures, and bouillon cultures from which the bacilli have been removed, are active, as they contain the toxins which have been secreted by the bacilli. These are soluble in water and can be precipitated by sodium-ammonia sulphate (Buchner) or zinc chlorid (Brieger and Boer). Dry preparations of the toxins which are more useful for experimental purposes may be made from the precipitate. According to Ehrlich and Madsen there are two toxins. They found in bouillon cultures *tetanospamin*, which has a strong affinity for nervous tissues and produces the muscular *spasms*, and *tetanolysin*, which dissolves red blood corpuscles.

The strength of the toxin is dependent upon the virulence of the bacilli. The virulence of the bacilli is remarkably increased by symbiosis with other bacteria, especially by putrefactive processes in the wound (A. Schütze).

In order to demonstrate the bacilli in a wound the penetrating foreign body or a particle of dirt should be transferred to an experimental animal. If bacilli are present, the symptoms of tetanus, which proves fatal after a few days, develop, and the spore-bearing bacilli can then be demonstrated microscopically in the wound secretion and can be obtained in pure cultures.

Tetanus a Wound Infection.—Tetanus is essentially a wound infection, even if clinicians are accustomed to differentiate a traumatic tetanus (with a demonstrable infection atrium) from a rheumatic or idiopathic tetanus (without a demonstrable infection atrium). Any injury of the epithelium of the skin or mucous membrane, however insignificant, may be followed by tetanus. Frequently the wounds which are followed by the disease are those in which the tissues are lacerated and contaminated with earth. It follows most frequently compound fractures, injuries produced by the explosion of a bomb, or by penetrating foreign bodies (for example, a splinter of wood); more rarely gangrenous wounds, scratched acne pustules, insect bites, and the wound resulting from the separation of the cord in the newborn (tetanus neonatorum). It sometimes follows lesions of the inner surface of the uterus, as in puerperal tetanus, and injuries of the epithelium of the mucous membrane of the nose and pharynx (supposedly in idiopathic tetanus).

Characteristics of Wounds Favoring Development of Tetanus Bacillus.—It is remarkable, considering the wide distribution and resistance of the bacilli, which have remained virulent for eleven years on a splinter of wood, that the disease is so rare. As a rule, the infection of the wound with tetanus bacilli is not alone enough to cause the disease. The bacillus demands special conditions for its development. Saprophytic organisms, usually found in wounds contaminated with earth or foreign bodies, favor the development of the bacilli, which, as a rule, are easily destroyed by the bactericidal substances in the tissue fluids. Severe injuries to the tissues (lacerations and contusions), which are followed by necrosis and putrefactive changes, also favor the growth of the bacilli. ["Necrotic tissue favors the proliferation of tetanus bacilli in two ways. In the first place, it seals up the wound to a certain extent, and thus provides the requisite anaërobic condition; in the second place, it would seem to prevent phagocytosis of the bacilli in some obscure way. It has been suggested that the strong chemotactic relation which exists between necrotic material and leucocytes causes the latter to take up dead tissue rather than bacilli. That innocent

foreign material may favor the development of tetanus was shown by Vaillard and Rouget. They demonstrated that tetanus would develop in the presence of an artificially produced hæmatoma or a subcutaneous fracture, while in the absence of such predisposing factors the bacilli were taken up by phagocytes."—Ricketts, " Infection, Immunity, and Serum Therapy," pp. 247 and 248.] Rational wound treatment (open treatment and drainage) often prevents the development of those conditions which favor the growth of the bacilli and the later development of the disease.

Epidemics of Tetanus.—Epidemics of tetanus have been observed in wars. These epidemics are easily explained, as all the important factors (severe injuries, contamination of the wound with street dirt or earth) which favored the infection of the wound and growth of the bacilli were present.

Post-operative tetanus, which often became endemic in earlier times, is only occasionally seen by the surgeon at the present time. In these cases the wounds are infected by soiled dressings, unclean instruments, etc. Tetanus has occasionally developed after gynecological operations (Koch, Phillips).

Tetanus bacilli rarely extend beyond the primary wound. They have been demonstrated in the neighboring lymphatic nodes (Schnitzler), in the viscera (Creite), in the circulating blood (Hochsinger), and in the blood taken from cadavers (Hohlbeck).

Incubation Period of Tetanus.—The incubation period in man varies from twenty-four hours to sixty days. As a rule, the disease develops between the eighth and fourteenth days. Tetanus has developed in four days after an accidental infection with a pure culture of the bacilli. [" In the statistics of Rose, twenty per cent of the cases showed symptoms in the first week, forty-five per cent in the second, and about thirty per cent in the third or fourth weeks."—Ricketts, " Infection, Immunity, and Serum Therapy," p. 249.] A certain time is required for the development of the bacteria and their toxins before they are absorbed and act upon the nervous tissues.

Condition of Infected Wounds.—There are no changes in the wound which are characteristic of an infection with tetanus bacilli. The wound may be suppurating, granulating, or healed when the first symptoms of the disease develop. Frequently foreign bodies on which the bacilli have gradually developed are found in the cicatrix.

Symptoms and Clinical Cause.—The chief symptoms of tetanus are tetanic muscular contractions accompanied by clonic spasms of greater or less degree, recurring at varying intervals. The muscular contraction is most pronounced in the muscles of mastication, and causes the painful " lock-jaw," the so-called trismus. More rarely the contrac-

tion begins in the muscles about the wound and then extends, so that later almost the entire musculature is involved. A high fever may be present from the beginning, or may develop later during the last hours or days of the disease. Fever may be absent in the fatal cases as well as in the cases which recover.

Tetanus pursues an acute or chronic course, depending upon the severity of the symptoms.

In the acute form, spasms of the muscles of the neck and face develop soon after the trismus. Then the muscles of the back, abdominal wall, and extremities become involved in frequently recurring clonic spasms. Unless the local spasms begin in the muscles of the arms, the latter are either spared or but little involved. Contraction of the muscles of the face gives the patient a characteristic grinning expression (risus sardonicus) and a senile appearance (facies tetanica) due to the wrinkling of the skin of the forehead and cheeks.

The painful muscular spasms, mostly tonic in character, may finally involve practically all the muscles of the body. These spasms, recurring at irregular intervals and lasting for different lengths of time, are often produced by the slightest irritation, such as touching the patient or by some noise, and so disturb him that sleep and the taking of food are rendered impossible. The attempt to swallow may bring on a convulsion, as the reflex excitability is so increased.

Bathed in sweat, with anxious expression and grinning mouth and with teeth pressed firmly against one another, the unfortunate patient awaits these frightful convulsions, which, if the extensor muscles of the back are involved, often force the head far back into the pillow (opisthotonos). The discharge of fæces and urine may be rendered difficult or impossible by the contraction of the sphincter muscles. If the muscles of respiration are involved, death from suffocation may occur twenty-four hours after the trismus. Spasm of the glottis, cardiac paralysis, and aspiration pneumonia may cause death. Shortly before death the temperature, which may be very high (109°–110° F.), falls. These excessively high temperatures are partly due to muscular action.

Prognosis.—Most frequently these acute cases terminate fatally within the first four days. Each day which the patient survives gives a better prognosis, for usually after a week the convulsions become less frequent and less severe, some groups of muscles lose their rigidity and are spared when subsequent spasms recur. The earlier the symptoms of tetanus develop after an injury, the more frequently the spasms recur and the more extensive the muscle groups involved, the graver the prognosis. [" In man, as in animals, it is found that the shorter the incubation period, the more severe the disease and the worse the prognosis. It is stated that of those cases where the incubation period is

under ten days, not more than 3 to 4.5 per cent recover; when the incubation period is from eleven to fifteen days, 25 per cent recover; in those cases in which the incubation period is still longer, about half the patients attacked throw off the disease. Different authors give different statistics, but these are the general results.''—T. C. Allbutt, '' System of Medicine,'' p. 773.] If acute symptoms do not develop again and if no complications, such as aspiration pneumonia, occur after improvement begins, the patient may slowly recover. Disappearance of the trismus and lessened excretion of sweat are indications of recovery, but both are unreliable.

In chronic forms this severe clinical picture is not seen. The muscles of respiration are not involved, and but little fever, or none at all, accompanies the difficulty in swallowing, the trismus, and the rigidity of the muscles of the neck. These may be the only symptoms. Recovery may occur after a week, at latest after three months.

Diagnosis.—The diagnosis, when the symptoms are pronounced, is easy. If, in the chronic forms, trismus is the only symptom at the beginning, all acute diseases of the mouth and pharynx which are associated with trismus and an elevation of temperature must be excluded.

Varieties of Tetanus.—*Tetanus in the newborn* (tetanus neonatorum) develops in from one to five days after the separation of the cord. The demonstration of bacilli in the pus discharged from the suppurating navel proves conclusively that the infection occurs here. The child presents the ordinary symptoms of the disease. The way in which the crying child releases the nipple, which was eagerly grasped, is rather striking. In most cases death occurs on the third or fourth day.

Puerperal Tetanus.—In puerperal tetanus (tetanus puerperalis) the infection is introduced by filthy midwives, often in performing abortions. The uterine mucous membrane affords the infection atrium for the bacilli which have been demonstrated in these cases. This form of tetanus is severe and ends fatally.

Head Tetanus.—Head tetanus (tetanus cephalicus) follows injuries in the area of distribution of one of the cranial nerves. The disease has received a number of names, selected because of its principal symptoms. In the acute severe forms pharyngeal and laryngeal spasms, the result of increased reflex excitability, soon develop. These resemble the spasms occurring in hydrophobia, and for this reason the disease has been called *tetanus hydrophobicus* by Rose. It is characterized by tetanic contractions of the muscles of mastication, combined with a paralysis of some of the muscles supplied by cranial nerves, particularly of those supplied by the facial nerve (therefore *tetanus facialis* according to Rose, or *tetanus paralyticus* according to Klemm). The rigidity then

extends to the muscles of the neck, trunk, and extremities. Death is produced by suffocation during a convulsion, or by cardiac paralysis.

In the subacute and chronic cases the symptoms are often mild, and may be limited to the region supplied by cranial nerves. According to Brunner, in some cases a tonic contraction of the muscles supplied by the facial nerve develops first upon the side of the injury, or, if the injury is in the median line, upon both sides; then follow immediately spasms of the muscles of mastication. In other cases, however, the muscles supplied by the facial nerve become paralyzed first upon the side of the injury, and spasms of the muscles of mastication then develop. It is remarkable that the paralysis never involves the motor branch of the fifth cranial nerve. Paralysis of the third and fourth cranial nerves has been noted when the injury involved the eye. As a rule, fever does not accompany head tetanus. Apparently the toxin extends along the nerves, injures the nuclei, and produces paralysis in this way.

Treatment.—In discussing the treatment of tetanus, serum therapy must be considered first. It is impossible to understand the action of antitetanic serum without a clear idea of the action of tetanus toxin, and therefore the mode of action of the latter will be briefly discussed.

After an incubation period varying with the animal used, a fatal disease follows subcutaneous and intravenous injections of tetanus toxins. It has been determined by experimental work that the toxins are absorbed from the wound by the end organs of motor nerves, and that they pass along the axis cylinders to the central nervous system. The toxins also circulate in the blood, from which they disappear when the spasms begin (Blumenthal). In all probability these toxins are deposited in some part of the neuromuscular apparatus. It has been shown that the toxins act upon the central nervous system, especially upon the motor centers of the spinal cord and medulla oblongata. The excitability of these centers is increased in tetanus, and any stimulus provokes a violent reaction. The results of the following experiments prove conclusively that the toxin does not act upon the muscles, the peripheral nerves, or brain (von Leyden and Blumenthal): (1) When the motor nerves are cut or the animal is curarized, the tetanic muscular contraction ceases; (2) when the cerebrum is removed, tetanus can still be produced (Brunner); (3) tetanic contractions do not develop in muscles when the corresponding spinal segments are destroyed.

Pathological Anatomy.—Microscopic changes in the motor ganglion cells have been described by Goldscheider, Flatau, and others. These changes are not regarded by other investigators as peculiar to tetanus. A. Wassermann and Takaki have shown that tetanus toxin has a strong

affinity for nervous tissue containing substances not found in other organs which bind the tetanus toxins. If a mixture of tetanus toxins and brain tissue is injected into guinea pigs or rabbits the animals do not develop the disease, as the toxins have already been rendered inert by their union with the nervous tissue. [" It is held by certain authors that the toxin attacks only the nervous tissue in man; in some of the lower animals, however, various organs, especially the liver, have an affinity for the toxin."—Ricketts, " Infection, Immunity, and Serum Therapy," p. 250.]

Absorption of Tetanus Toxin.—It has been demonstrated by the experiments of Meyer and Ransom, Tiberti, and others that the toxins pass along the peripheral (motor) nerves to the central nervous system. Local contractions which are not frequent in man, except in head tetanus, occur in animals after subcutaneous (not after intravenous) injections. These local contractions are due to the extension of the toxins along the nerves to the segments corresponding to the muscles about the wound (Stinzing and others).

Tetanus Antitoxin.—If an animal has recovered from the disease it is immune against small doses of virulent toxins or larger doses of attenuated toxins. By injecting gradually increasing doses of the toxins the immunity may be so raised that the animal can withstand the injections of pure tetanus toxins or of virulent bacilli. The animal has become immune, and, according to Behring and Kitasato, its blood serum has the power to neutralize tetanus toxin, to protect other animals against tetanus, and to cure them when the disease has already developed. The bacilli are not killed by the antitoxin, but they are no longer active, as their toxin is rendered harmless. The blood serum of immunized horses (antitoxin) affords a certain protection to man when used in the treatment of tetanus.

According to our present knowledge, a concentrated antitoxin is able to neutralize the tetanus toxin circulating in the blood, and if the organism is not flooded with large quantities of the toxin, one may hope by repeated injections not only to neutralize the toxin in the blood, but also to render harmless the toxins continually absorbed from the wound, before they can act upon the ganglion cells in the spinal cord and medulla. As soon as the toxin becomes united with the ganglion cells, the antitoxin circulating in the blood no longer has any effect, as it either reaches the spinal cord in too small amounts or is unable to break up the chemical union between the toxin and the ganglion cell. Attempts have been made to bring the serum into direct contact with the central nervous system and the centers upon which the toxins act. Jacob has injected the serum into the subdural space, Kocher into the ventricles of the brain. Experimental work indicates

that direct injection is of some value. No definite conclusions can be drawn from clinical experience.

Antitoxin should be injected as soon as possible after the first symptoms of the disease develop (according to von Behring within the first thirty hours), and a subcutaneous or intravenous injection of a certain amount should be made daily. Unpleasant symptoms follow the intravenous injections of some sera, and for that reason von Behring recommends that subcutaneous injections should be made, preferably in the area surrounding the wound. In tetanus puerperalis the serum should be injected into the vagina, in tetanus neonatorum into the abdominal cavity. Calmette recommends that the dried serum be sprinkled on the wound. Küster has exposed the nerves supplying the region of the wound and has injected the serum into them. This treatment has apparently been successful in some cases.

[Antitetanic serums are not standardized by American manufacturers, and it is impossible to control accurately the dosage. Not less than 10 c.c. should be given for prophylactic purposes, and this dose should be repeated. It is impossible to set any definite limits for the amounts which should be used for curative purposes. As previously mentioned the curative action of the serum cannot be relied upon. It is most useful as a prophylactic, and should be given in all cases in which there is a possibility that tetanus may develop.]

Technic for Injection of Serum into the Lateral Ventricle and the Spinal Subdural Space.—In Kocher's method of injecting into the lateral ventricle a small trephine opening is made in the skull from 1 to 1¼ inches lateral to the bregma, the point at which the sagittal and coronal sutures meet. A long needle is then passed 2 or 2½ inches into the brain substance in a vertical direction. When fluid flows from the needle, the serum is injected slowly. Tavel makes the opening 1¼ inches from the median line and 1¼ anterior to the coronal sutures. He then passes the needle toward the foramen magnum. The lateral ventricle may also be reached from the frontal region, from just above and a little to the inner side of the frontal eminence (von Bergmann), from the lateral surface of the skull (Keen), and from the occipital region (Beck). It is best to make a skin periosteal flap in exposing the area in which the opening is to be made. The flap can then be sutured in position and subsequent injections made through it (Tavel). The danger of infection is lessened by this procedure.

The technic employed in Quincke's lumbar puncture is used in making spinal injections. The patient is placed upon his left side and a needle is passed between the spines of the third and fourth lumbar vertebræ, and is forced forward somewhat upward and inward. After considerable cerebro-spinal fluid has escaped, the serum is injected slowly.

Results of Serum Treatment.—It is difficult to judge of the value of the serum treatment in mild cases, as a large proportion of these recover spontaneously. On the other hand, in the severe cases, which usually develop within a week after the injury, the serum has no curative action, although it has been demonstrated that it passes through the body, as it has been found in the urine (von Leyden).

As the serum treatment cannot be relied upon when the disease has developed, it is the duty of the attending physician to try any treatment which may possibly cure the disease or at least alleviate the suffering.

Treatment of the Wound.—Toxins are being continually absorbed from the wound, and this should be prevented. When the position and form of the wound permits, as in the fingers or toes, an amputation or a thorough excision should be performed. In extensive and complicated injuries of the extremities, particularly in compound fractures, amputation is indicated as soon as the first symptoms develop. Only in the mildest cases should this indication be disregarded. All necrotic tissue and blood clots should be removed from the large wounds of the trunk, and the undermined soft tissues should be opened widely in order to prevent putrefaction, which, according to experimental work, increases the virulence of the bacilli. The aseptic, open treatment of the wound is an important preventive measure, as it permits of the free access of air which prevents the growth of anaërobic bacilli. Foreign bodies, lying in the wound or encapsulated in the scar, should be removed, as large numbers of bacilli are usually attached to them.

Cauterization of the wound, which is frequently recommended by physicians, does harm, as the eschar which forms prevents the discharge of wound secretion. If the wound is contaminated with street dust, manure, or earth, or has been received in localities where tetanus is of frequent occurrence, a prophylactic injection of serum should be given, and if practicable the wound should be excised. In spite of such prophylactic injections, tetanus may develop and end fatally, even if the majority of cases handled in this way recover (Suter).

The remaining treatment is purely symptomatic. Narcotics should be given to control the spasms, especially the dangerous spasms of the muscles of respiration. Large doses of morphin and chloral act best. In feeding the patient, the dangers of aspiration pneumonia should be kept in mind. Rectal feeding is indicated as long as the trismus and pharyngeal spasms persist. Any external irritation will cause convulsions and the patient must be placed in quiet surroundings. Morphin should be given before the dressings are changed or the patient is catheterized. Stimulants should be given as the heart becomes weak.

LITERATURE.—*v. Behring* und *Kitasato.* Ueber das Zustandekommen der Diphtherie und der Tetanusimmunität bei Tieren. Deutsche med. Wochenschr., 1809, p. 1113.—*Bergell* und *Levy.* Ueber den Einfluss des Curare bei Tetanus. Therapie der Gegenwart, 1904, p. 396.—*Brunner.* Kopftetanus. Beitr. z. klin. Chir., Bd. 9, 10 u. 12.—*Calmette.* Sur l'absorption de l'antitoxin tétanique par les plaies. Académie des sciences, Mai, 1903.—*Creite.* Zum Nachweis von Tetanusbazillen in Organen des Menschen. Centralbl. f. Bakteriol., Bd. 37, Orig., 1904, p. 312.—*Hohlbeck.* Vorkommen des Tetanusbazillus ausserhalb der Infektionsstelle beim Menschen. Deutsche med. Wochenschr., 1903, p. 172.—*Kitasato.* Ueber den Tetanusbazillus. Zeitschr. f. Hygiene, Bd. 7, 1889, p. 225.—*E. Koch.* Tetanus nach Bauchoperat. Deutsche Zeitschr. f. Chir., Bd. 48, 1898, p. 417.—*Kruse,* in Die Mikroorganismen von Flügge, 1896, Bd. 2.—*Küster.* Ein Fall von örtl. Tetanus. Antitoxineinspritzungen in die Nervenstämme. Heilung. Chir.-Kongr. Verhandl., 1905, II, p. 161.—*Lexer.* Zur Tetanusbehandlung. Therapie. d. Gegenwart, 1901, Juni.—*v. Leyden* und *Blumenthal.* Der Tetanus. Spez. Path. u. Ther. von Nothnagel, V. Bd., 1900.—*v. Lingelsheim.* Tetanus. In Kolle-Wassermanns' Handb. d. pathog. Mikroorganismen, Bd. 2, 1903, p. 566, with Lit.—*Marx.* Ueber die Tetanusgift neutralisierende Eigenschaft des Gehirnes. Zeitschr. f. Hygiene u. Inf., Bd. 40, 1902, p. 231.—*Meyer* und *Ransom.* Untersuch. über d. Tetanus. Arch. f. experim. Pathol., Bd. 49, 1903, Part 6.—*Neumann.* Der Kopftetanus. Kritisches Sammelreferat. Centralbl. f. Grenzgeb., Bd. 5, 1902, p. 503.—*Nicolaier.* Ueber infektiösen Tetanus. Deutsche med. Wochenschr., 1884, p. 842.—*Philips.* Tetanus as a Complication of Ovariotomy. The Lancet, 1892, p. 139.—*Rose.* Trismus und Tetanus. Deutsche Chir.—*Rosenbach.* Zur Aetiologie des Wundstarrkrampfes. Arch. f. klin. Chir., Bd. 34, 1886, p. 306.—*Steuer.* Sammelreferat über die Therapie des Tetanus. Centralbl. f. Grenzgeb., 1900, Bd. 3.—*Stinzing.* Beitrag zur Lehre des Tetanus traumaticus. Grenzgeb. d. Med. u. Chir., 1898, Bd. 3 und Münch. med. Wochenschr., 1898, p. 1265.—*Suter.* Zur Serumbehandlung des Starrkrampfes, insbes. über Tetanuserkrankungen trotz prophylaktischer Serumtherapie. Arch. f. klin. Chir., Bd. 75, 1905, p. 113.—*Tiberti.* Ueber den Transpert des Tetanusgiftes zu den Rückenmarkszentren durch die Nervenfasern. Centralbl. f. Bakteriol., Bd. 38, Orig., 1905, p. 413.—*v. Töröck.* Experim. Beiträge zur Therapie des Tetanus. Zeitschr. f. Heilkunde, 1900, Bd. 21.

CHAPTER IV

DIPHTHERIA

THE so-called diphtheritic inflammation of the skin and mucous membranes is a fibrinous inflammation associated with extensive necrosis. This particular form of inflammation, sometimes superficial and sometimes deep, is not caused by diphtheria bacilli only. Typhoid and dysentery bacilli, streptococci and chemicals (ammonia) produce similar changes in mucous membranes. Diphtheria bacilli are the cause of the epidemic infectious disease called diphtheria in which the mucous membranes, especially those of the upper part of the respiratory and alimentary tracts, are inflamed. Streptococci and staphylococci are frequently associated with the diphtheria bacilli in these cases, or they alone may produce a fibrinous inflammation and necrosis of the mucous

membranes, such as frequently occur in scarlet fever (diphtheroid scarlatina).

Bacillus of Diphtheria.—Diphtheria bacilli, which were first obtained in pure cultures by Löffler (1884), are to be regarded as the cause of epidemic diphtheria, and occasionally of wound diphtheria. They are slender, often somewhat curved, rods, usually lying irregularly scattered, frequently in clusters. The bacilli are not motile, are about as long as the tubercle bacillus and slightly broader. Often one end is thickened, but they do not form spores. They are found in the membranes covering mucous surfaces, in pneumonic foci in patients dying of diphtheria, and have also been demonstrated in the adjacent lymph nodes, in the blood of cadavers, in the pus from submucous phlegmons, and in metastatic abscesses and in wounds.

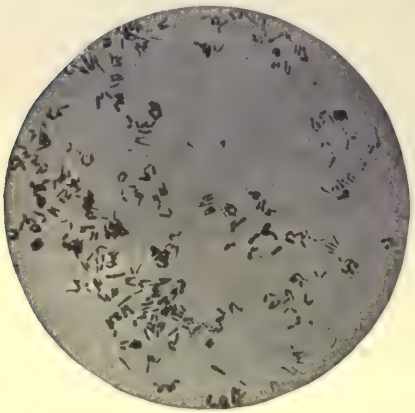

Fig. 135.—Diphtheria Bacilli.

They stain with Löffler's alkaline methylene-blue solution, Ziehl's carbol-fuchsin, and with Gram's method.

They grow best upon Löffler's solidified blood serum, also upon glycerin-agar, with free access of air. Pinhead size, whitish gray, opaque colonies appear after twenty-four hours. The borders of the colonies, when viewed under a glass, are irregular and slightly granular, and may readily be distinguished from the small and transparent colonies of streptococci growing near by. In bouillon they form small granules, which become attached to the test tube.

For diagnostic purposes a piece of the membrane should be removed with sterile forceps, washed in sterile water to remove the bacteria of the mouth, and a number of stroke cultures made upon slant media. After ten hours at the earliest, grayish yellow streaks appear upon the surface, from which microscopic preparations may be made. The bacilli should be stained according to Gram's method. Animal experiments should also be made to prevent mistaking them for the non-pathogenic pseudo-diphtheria bacilli, similar in appearance and found in the mouth.

Guinea pigs are best suited for experimental purposes; usually 0.5 c.c. of a twenty-four-hour old bouillon culture will kill these animals. Rabbits, sheep, young dogs, cattle, horses, hens, pigeons, and cats are susceptible; mice and rats are not. After subcutaneous injections of

cultures, animals die in twenty-four hours or in from one to two weeks, depending upon the virulence of the bacteria and the number injected.

Œdema about the point of injection, pleural exudates, and fatty degeneration of the viscera are found when a post-mortem examination is made. Paralyses have been observed. Infections of the mucous membrane produce an inflammation associated with necrosis.

Subcutaneous injections of filtered bacteria-free cultures produce the same results, as they contain the poisonous metabolic product (toxins) of the bacilli. Diphtheria bacilli are therefore, like tetanus bacilli, toxic bacteria. The infection is primarily a local one, and the general symptoms are produced by the absorption of the toxins. Only rarely do the bacilli pass beyond the primary focus of infection.

According to the investigations of Brieger and C. Fränkel, diphtheria toxin should be regarded as a toxalbumin; but when pure it does not respond to the tests employed for either albumins or peptones (Brieger and Boer). Optically, it is inactive and cannot be placed in any of the groups known to organic chemistry (Beck).

Diphtheria Antitoxin.—The blood serum of experimental animals immunized against diphtheria protects other animals from infection and cures those in which the symptoms have already developed (von Behring and Wernicke). It contains an antitoxin. Diphtheria bacilli, like tetanus bacilli, are not killed by the antitoxin, but their toxins are neutralized and the bacilli gradually disappear from the body. The blood serum of patients who have recovered from the disease has for a short time the power of immunizing animals (Klemensiewicz and Escherich, Abel).

The serum used in the treatment of the disease is obtained almost exclusively from horses (von Behring) (*vide* Treatment).

Modes of Infection and Susceptibility.—Infection in man follows direct or indirect transference of the bacilli from a patient. Direct infection may follow kissing, coughing, and sneezing. Infection may be carried by any object, especially eating utensils and handkerchiefs which have come in contact with the patient's mouth or the secretions from his mouth and nose. Diphtheria bacilli may cause a febrile angina without a membrane. Convalescent patients, and even healthy people who have been about diphtheria patients, harbor bacilli in their mouths, and so it is possible for the disease to be transferred by people who are not sick. The patient's excreta are especially dangerous, as the bacilli remain viable from three to four months in the dried condition; for example, in the expectorated and dried membrane.

All people are not, however, susceptible to the disease. Children from two to four years of age are most, adults least, susceptible. Among adults immune persons are found whose blood serum has a pro-

tective action, although they have never had the disease (Abel, Wassermann). Diseases of the mucous membranes, chronic catarrh and angina, such as occur in measles and scarlet fever, favor the development of diphtheria. An immunity, which lasts for a short time, follows the disease.

It is doubtful whether the disease is transferred from animals to man.

DIPHTHERIA OF MUCOUS MEMBRANES

Löffler's bacillus, usually associated with other bacteria, produces an inflammation which involves most frequently first the mucous membrane covering the tonsils, the pillars of the fauces, and the pharynx; and which then may extend to the mucous membranes of the nose, larynx, trachea, and finer bronchi, and the middle ear. Much more rarely the inflammation involves primarily the mucous membrane of the larynx and nose. Primary inflammation of the mucous membrane of the vagina and secondary involvement of the œsophageal and gastric mucous membranes occur, but are very rare.

Onset.—The disease begins with general and local symptoms. Often it develops suddenly with high fever, delirium, etc.; often slowly with prostration, chilly feelings, and loss of appetite. The first local symptoms naturally depend upon the location of the infection. Diphtheria of the pharynx begins with pain upon swallowing; of the larynx, with hoarseness, coughing, and dyspnœa; of the nose, with a profuse, purulent, hæmorrhagic discharge.

At first the infected mucous membrane is swollen, glistening, and reddish. Soon small, whitish, slightly raised patches appear (in pharyngeal diphtheria these appear upon the tonsils first). At first these patches may be easily removed, as they are composed of fibrin only, which is deposited upon the sloughing epithelium. These patches gradually extend and become thicker as an exudate is poured out, so that, for example, in pharyngeal diphtheria after a few days the tonsils, the pillars of the fauces, the uvula, and the entire pharyngeal wall become covered with a whitish or grayish yellow membrane. The inflammation may extend deeper and involve the connective tissues of the mucous membrane, then these become infiltrated and necrotic.

Pseudo-membrane.—In the beginning of the disease and in mild cases the pseudo-membrane is but loosely attached to the surface of the epithelium. When removed only the epithelial cells are taken with it, and healing without scar formation may occur if a new membrane does not form. In advanced and severe cases the connective tissues of the mucous membrane are also involved, and the membrane is often removed with difficulty, and when removed leaves bleeding surfaces. It is no

longer correct to distinguish in epidemic diphtheria between croupous and diphtheritic changes or between croup [1] and diphtheria,[2] for they are only different phases of the same local pathological processes. As a rule, the pseudo-membrane is more closely attached to squamous than to ciliated epithelium, as in the former there is no basal membrane to prevent its attachment to the underlying connective tissues.

After spontaneous separation of the pseudo-membrane, the mucous membrane almost always heals without a scar. On the other hand, when the pseudo-membrane is forcibly removed, another membrane usually forms and the necrosis extends deeper. Scars form in the tonsils only when the tissues of the same undergo extensive necrosis.

Separation of Pseudo-membrane.—The separation of the membrane occurs much more rapidly in adults than in children. In the former it may begin after the first day, while in the latter after the first week (Rumpf).

Extension the Result of Secondary Infection.—The inflammation rarely extends to the cartilages of the larynx and the bones of the nose. The extension in the severer cases is due to secondary infections with other bacteria (putrefactive or pyogenic). The fibrinous necrotic areas then undergo putrefactive changes or a gangrene of the entire mucous membrane develops. An extensive cicatricial stenosis of the larynx may follow these secondary infections.

In diphtheria infections there is an inflammatory œdema of the submucous tissues, which occasionally ends in suppuration (abscess, phlegmon). Pyogenic bacteria are important factors in abscess formation, but diphtheria bacilli alone have been found in the pus (Tavel).

The adjacent lymphatic nodes are always swollen and inflamed. Frequently they contain small grayish white necrotic foci, but abscesses rarely develop.

According to Frosch, bacilli may be much more frequently demonstrated in the blood and viscera than was formerly considered to be the case.

Histology of Lesions.—Microscopically (Fig. 136) there is found in freshly inflamed areas a layer of fibrin, the fibers of which are arranged in meshes which cover the degenerating epithelium. The meshes of this fibrin layer contain degenerated epithelial cells and leucocytes. Its

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[1] A fibrinous inflammation, which is not due to Löffler's bacillus, occurs in the pharynx and larynx in a number of different infectious diseases, such as the acute exanthemata, typhoid fever, whooping cough, and pneumonia. This is called secondary croup, or better, diphtheroid.

[2] The term diphtheritis is used by many to designate the local changes which occur in epidemic diphtheria. There is, however, at the present time, no uniformity of opinion concerning the use of this term.

finer fibers extend down to the inflamed and infiltrated tissues. Older membranes are stratified, the oldest and most superficial layers consisting of layers of epithelial cells and fibrin, and containing large numbers of saprophytes. Then follows a fine-meshed and then a more recent coarse-meshed layer of fibrin. The bacilli are usually found in the first layer, while the coarse-meshed layer is firmly attached to the inflamed and infiltrated connective tissue.

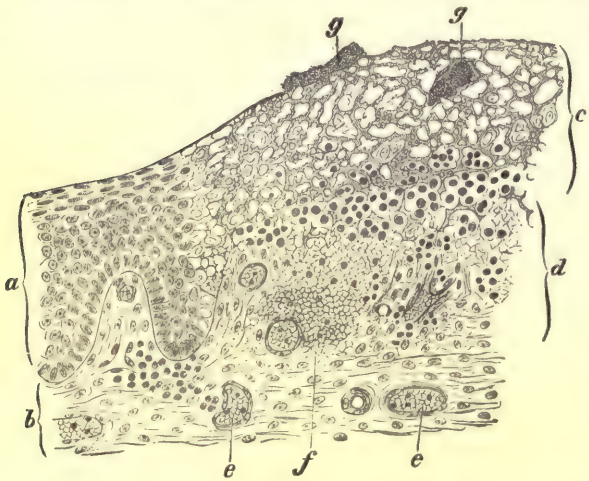

FIG. 136.—SECTION THROUGH THE UVULA, FROM A CASE OF PHARYNGEAL DIPHTHERIA. (After Ziegler.) *a*, Normal epithelium; *b*, submucous connective tissue; *c*, fibrin with a netlike arrangement; *d*, fibrin infiltrated with round cells lying upon necrotic connective tissue; *e*, blood vessels; *f*, a hæmorrhagic focus; *g*, groups of micrococci.

Clinical Course and Severity of the Disease.—The clinical course of the disease is usually so acute that recovery or death occurs within a week.

The severity of the disease depends upon the character of the epidemic, the position and extension of the inflammation, and upon mixed infections. Laryngeal is much more dangerous than pharyngeal diphtheria, and the prognosis is very unfavorable if secondary infection with other bacteria, especially streptococci, occurs.

Prognosis.—The mortality is especially high in children. Almost all children in the first year of life die. The mortality decreases as age advances.

Causes of Death.—There are a number of causes of death, depending upon the local course of the disease, the general toxic infections, and complications.

The inflammation of the mucous membrane alone is frequently the cause of death in young children, as the membrane, the profuse secretion, and the resulting œdema produce a rapidly developing stenosis of the larynx. Suffocation may be prevented by tracheotomy. Tracheotomy has a favorable influence upon the course of the disease only when it is performed early, before a marked asphyxia has developed, and when the disease remains limited to the larynx. If the inflammation extends to the bronchi, tracheotomy will not save the patient, for the fibrinous masses fill the bronchi and their branches, and suffoca-

tion occurs, even if occasionally large masses of the membrane are coughed up.

The general toxic infection causes changes in the nerves supplying the heart or in the heart muscle. Death often follows a few days after the development of the disease, often during convalescence, from paralysis of the vagus, fatty degeneration of the heart muscle, or paralysis of the cardiac ganglia.

Complications.—A number of complications which are not fatal are caused by the toxins, such as diseases of the kidneys (albuminuria and acute nephritis) and the diphtheritic paralysis, which apparently is an ascending neuritis with subsequent central degeneration (Baginsky, Rainy). An early paralysis of the soft palate developing in severe cases is differentiated from a late paralysis developing usually in the second and third week, which recovers slowly but spontaneously. The muscles of the palate and pharynx with disturbance of speech and swallowing, the extrinsic muscles of the eye, and the muscles of accommodation are most frequently paralyzed; the muscles of the face, vocal cords, diaphragm, trunk, and extremities more rarely. Hemiplegia may be caused by cerebral hæmorrhage or embolism.

BRONCHO-PNEUMONIA, due to aspiration or extension of the inflammatory process to the lungs, and MIXED INFECTIONS, particularly with streptococci, are to be regarded as serious complications. Not only the local changes, necrosis of the epithelium, and phlegmonous inflammation, but also the general symptoms, become more severe in poly-infections. Then, usually, the clinical picture of a general putrefactive infection rapidly develops. The inflamed mucous surface becomes covered with a dirty, blackish membrane, and a foul-smelling secretion is discharged. There may be some fever or the temperature may be subnormal, the patient rapidly fails, the heart becomes weak, hæmorrhages occur into the skin, the joints become inflamed, endocarditis and nephritis may develop (putrid diphtheria). The toxins secreted by putrefactive bacteria and streptococci are important factors in these cases, as the frequent demonstration of streptococci in the blood and the not infrequent inefficiency of the antitoxin show. Such cases almost always end fatally within a few days.

Diagnosis.—The diagnosis of diphtheria is not difficult in acute cases when the pathological changes in the mucous membranes are visible. It may be mistaken for a follicular angina. In laryngeal diphtheria without pharyngeal involvement, the examination of the expectorated membrane and the laryngoscopic findings exclude other forms of inflammation. In adults it may be confused with syphilis of the tonsil.

In all cases diphtheria bacilli should be demonstrated by cultural methods. If the pharynx is wiped with a small piece of sterile gauze

or with an applicator, and a number of stroke cultures are made, a definite diagnosis can be made, after a little practice, in twelve hours.

Treatment.—It is most important in treating the local condition that chemical or mechanical irritation which might favor the extension of the inflammation and the absorption of toxins be avoided. In fact, the great number of agents (caustics, antiseptics, emetics for mechanical removal of the membrane) which have been recommended in the treatment indicate of how little value they have been.

On the other hand, salt solution, frequently inhaled, and mild antiseptic gargles have a very favorable action upon the inflamed mucous surface and favor the separation of the membrane.

Antitoxin is of the greatest importance in the general treatment.

[The value of antitoxin, both for prophylactic and curative purposes, has been demonstrated. The amount used for curative purposes depends upon the virulence of the infection and the time at which the patient is seen. The average dose recommended by the United States Pharmacopœia is 3,000 units. The Chicago Health Department advises that from 3,000 to 8,000 units be given in ordinary cases. From 1,000 to 1,500 units should be given when the patient is first seen, and the injection may be repeated if there is no improvement within twenty-four hours. In the severe cases, 8,000, 10,000, and 14,000 units have been given, and the patients have not suffered from such quantities. The serum may be injected under the skin of the thorax, thigh, or back. The earlier the serum is injected the better the results will be.]

Prophylactic injections of from 200 to 500 units are to be recommended in epidemics.

Results of Antitoxin Treatment.—In most cases a marked improvement is noted soon after the injection. The inflammation extends no farther, the membrane becomes loosened, the symptoms of stenosis subside, the swelling of the mucous membrane disappears, the general condition improves, and the fever falls.

The mortality has been greatly reduced since the serum treatment has been employed. Antitoxin has caused a reduction of more than fifty per cent in the mortality (Ricketts); from forty-one per cent to eight or nine per cent (Baginsky).

After several days eruptions which resemble those of urticaria and measles, also swelling of the joints, frequently follow the injections.

In the severe forms, in which gangrene and secondary streptococcic infections develop—the so-called putrid diphtheria—antitoxin frequently gives no results.

The remaining treatment should attempt to control the symptoms and complications (cardiac weakness, paralysis, nephritis, phlegmons, cicatricial laryngeal stenosis) as they develop.

DIPHTHERIA OF THE SKIN

Not infrequently the edges of tracheotomy wounds become gangrenous. Later these wounds suppurate and healthy granulation tissue develops. Sometimes, however, the gangrene extends to adjacent and deeper tissues; the trachea and both sterno-cleido-mastoid muscles may then become exposed and large defects in the anterior wall of the trachea may develop.

This acute progressive gangrene of wounds may be caused by diphtheria bacilli. When these bacilli gain access to a wound they produce a coagulation necrosis of its surfaces and a severe inflammation of the surrounding tissues. The surface of the wound first becomes covered with a dirty, grayish red, firmly adherent membrane, and later the tissues become necrotic and gangrenous. The necrosis and gangrene may extend beneath the edges of the wound.

General symptoms, if present, are the same as those accompanying diphtheria of mucous membranes; even paralysis has been observed (Billroth). According to Billroth, wound diphtheria was of fairly frequent occurrence in pre-antiseptic times in hospitals for children; and in severe epidemics all possible forms of accidental- and operation-wounds were attacked.

It is rarely seen at the present time, except in tracheotomy wounds in patients sick with diphtheria. The bacteriological investigations of Brunner and others have shown, however, that the earlier observations as to the diphtheritic nature of these infections were correct. It has also been demonstrated that diphtheria bacilli may produce a diphtheritic inflammation without general symptoms, an inflammation with a fibrinous membrane (Schottmüller and others), or, associated with pyogenic bacteria, suppuration (Brunner, Tavel, and others).

The diagnosis of mild forms of wound diphtheria is very difficult. A grayish white or yellowish membrane frequently develops upon granulation tissue which has been infected with streptococci, staphylococci, the bacillus pyocyaneus, etc., and it is often difficult to differentiate between these infections and those due to diphtheria bacilli. The more severe infections resemble noma and hospital gangrene, which are rarely seen at the present time. A bacteriological examination is important in all cases and will determine the diagnosis.

In the local treatment of wound diphtheria—general treatment is required in only the severest cases—all agents which injure the wound surfaces should be avoided, and the separation of the diseased tissue and the formation of healthy granulations should be favored by the use of moist dressings.

It is important to prevent the development of diphtheria in trache-

otomy wounds. After the tube is introduced, the wound should be lightly packed with iodoform gauze to protect the fresh surfaces from infection from the trachea until healthy granulation tissue forms.

The first indication in the treatment of diphtheria patients—who, because of the dangers of transmitting the infection, should always be kept in isolation wards—is to render harmless the toxins secreted by the bacilli. It is to the lasting credit of von Behring that he has given us not only the fundamental principles of immunity against infectious diseases, but also a serum which cures diphtheria.

Literature.—*Baginsky.* Diphtherie und diphtheritischer Krupp. Wien, 1898, Nothnagel's Handb., Bd. 2, and Deutsche Klinik, Bd. 2, 1903.—*M. Beck.* Diphtherie. In Kolle-Wassermann's Handb. d. pathog. Mikroorgan., Bd. 2, 1903, p. 754. —*v. Behring.* Die experimentelle Begründung der antitoxischen Diphtherietherapie. Deutsche Klinik, Bd. 1, 1903, p. 73; Diphtherie. Bibl. v. Coler, Bd. 2.—*Brunner.* Ueber Wunddiphtheritis. Berlin. klin. Wochenschr., 1893, p. 515; Eine weitere Beobachtung von Wunddiphth. Ibid., 1894, p. 310; Wundinfektion u. Wundbehandl., Frauenfeld, 1898, II, p. 130.—*Cohn.* Erfahrungen über Serumbehandl. d. Diphtherie. Mitteil. aus den Grenzgebieten, Bd. 13, 1905.—*Ehrlich.* Ueber die Konstitution des Diphtheriegiftes. Deutsche med. Wochenschr., 1898, p. 597.—*Eröss.* Ueber d. Mortalität d. Diphtherie. Jahrb. f. Kinderheilk., III. Folge, Bd. 10, 1905, p. 595.—*Freymuth* und *Petruschky.* Vulvitis gangrænosa mit Diphtheriebazillenbefund. Deutsche med. Wochenschr., 1898, p. 232.—*Gottstein.* Die Periodizität der Diphtherie u. ihre Ursachen. Berlin, 1903.—*Günther.* Bakteriologie. Leipzig, 1902.—*Heim.* Bakteriologie. Stuttgart, 1898.—*Krönlein.* Ueber die Resultate der Diphtheriebehandlung mit besonderer Berücksichtigung der Serumtherapie. Chir.-Kongress, Verhandl., 1898, I.-S., 105.—*Kruse,* in Die Mikroorganismen von Flügge, Bd. 2, Leipzig, 1896.—*Vowack.* Blutbefunde bei an Diphtherie verstorbenen Kindern. Centralbl. f. Bakteriol., Bd. 19, 1896, p. 982.—*Rumpf.* Diphtherie. Handb. d. praktischen Medizin, Stuttgart, 1901, Bd. 5.—*Rainy.* On the Action of Diphth. Toxin on the Spinal Stictochrome Cells. Journ. of Path. and Bact., 1900, p. 612.—*Schottmüller.* Wunddiphtherie u. s. w. Deutsche med. Wochenschr., 1895, p. 272.—*Tavel.* Ueber Wunddiphtherie. Deutsche Zeitschr. f. Chir., Bd. 60, 1901, p. 460; Diphtherie. In Kocher's chirurg. Enzyklopädie.—*Wieland.* Das Diphtherieheilserum. Jahrb. f. Kinderheilk., Nr. 7, Bd. 7, 1904, p. 527.—*Wright* und *Emerson.* Ueber das Vorkommen des Bak. diphth. ausserhalb des Körpers. Centralbl. f. Bakteriol., Bd. 16, 1894, p. 412.

CHAPTER V

ANTHRAX

Davaine in 1863 recognized that bacteria were the cause of the disease, and originated the name bacillus anthracis. The bacilli cultivated and described by Koch in 1876 are slender, cylindrical, non-motile rods from 6 to 10 μ in length. They are often united in tissues and culture media to form long chains.

The Bacillus of Anthrax.—The bacilli stain with aniline dyes, hæmotoxylin, and by Gram's method. A capsule surrounds the bacillus when growing in the body, but it is difficult to obtain it in culture media. If present, the capsule appears in stained preparation as a narrow, clear zone about the bacillus. Clear oval spaces, which correspond to the spores, are seen in bacilli in stained preparation. Often each member of a long chain contains a spore, which is set free when the bacillus degenerates. When young bacilli grow on fresh media they swell, then rupture at one end and discharge the spore. Anthrax spores, because so resistant, are especially suited for testing the efficiency of different methods of sterilization. They are destroyed by live steam (212° F.) in five minutes; dry heat, on the other hand, even of 284° F., must act for a longer time (three hours or more).

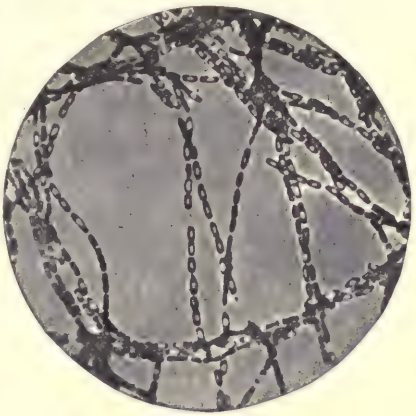

FIG. 137.—ANTHRAX BACILLI.

Method of Staining Spores.—Klein's method is best suited for staining anthrax spores. In this method, equal parts of a physiological salt solution suspension of material containing anthrax spores and Ziehl's carbol-fuchsin are mixed in a watch crystal or glass. This mixture is heated until steam arises; then a few drops are transferred to a cover glass. After it is dried it is destained with a one per cent solution of sulphuric acid, washed with sterile water, and counter-stained with dilute methylene blue for three or four minutes (Sobernheim).

Cultural Characteristics.—The growth of anthrax bacilli upon gelatin is very characteristic; upon agar (with free access of oxygen) less so. The borders of the colonies, which appear as dark gray points, have a wavy or wreathed appearance, while in stab cultures numberless processes grow out from the needle track, like the bars of a feather.

Effects of Symbiosis with Other Bacteria.—The bacilli rapidly die in mixed cultures with the bacillus pyocyaneus. The latter produces a fermentlike substance (pyocyanase) which dissolves anthrax bacilli (Emmerich). Streptococci and staphylococci are antagonistic, especially so in the body.

Anthrax in Experimental Animals.—White mice, guinea pigs, and rabbits are best suited for experimental purposes. These animals die in from one to three days after the infection of small cutaneous wounds with spore-free and spore-containing bacilli. Large numbers of bacilli

are found in the blood vessels of the viscera. According to Schimmelbusch's experiments, the absorption of the bacilli is so rapid that in mice the amputation of the tail, a wound of which has been infected, does not prevent death, even if performed as early as ten minutes after the infection. After a half hour, bacilli may be demonstrated in the viscera. Animals die rapidly of a general infection after bacilli are rubbed into an intact skin (Wasmuth). Intestinal ulcers and fatal general infections follow feeding experiments with spore-containing bacilli. Spore-free bacilli are killed by the gastric juice and cause no symptoms. Infections of the respiratory tract follow the inhalation of infected dust (Buchner, Enderlen).

Neither toxins nor endotoxins have been demonstrated. The toxic albuminous substances found in the blood and viscera by Hoffa and others are not to be regarded as specific anthrax toxins, but as toxic decomposition products (Sobernheim).

Occurrence of Anthrax Bacilli Outside of the Body.—Anthrax spores are discharged upon the surface of the ground in the excreta of diseased animals. They remain viable in damp places for a long time (two to three years), and are widely distributed in all possible ways by animals and man, rains, floods, etc., over meadows and pasture lands. Grazing animals (cow, sheep, horses) ingest the spores with their food, and for this reason almost always develop the intestinal form of anthrax. Primary anthrax of the lung does not occur in these animals; anthrax of the skin but rarely. Other animals (rats, dogs, and pigs) are immune.

Immunization of Susceptible Animals.—Susceptible animals may be immunized by the injection of attenuated cultures (Pasteur's protective inoculation). The blood serum of immunized animals has protective and curative properties, especially if, as Sobernheim demonstrated, the serum and attenuated cultures of the bacilli are injected simultaneously (mixed active and passive immunization). The serum treatment in man was first successfully employed by Sclavo and Mendez. [The best known serums are those of Sclavo, prepared from the goat and ass, and those of Mendez and Deutsch. The properties on which the value of the serums depends are unknown. Sobernheim is very positive in stating that the bactericidal power of the animal's serum is not increased by immunization or infection, and the existence of an antitoxin is not recognized. As in some other instances, immunization may cause an increase in the opsonins which would render the serum effective by its power to cause increased phagocytosis.

The method of Sobernheim, that of mixed active and passive immunization, seems to be successful as a prophylactic measure. The vaccine consists of a mixture of antiserum and bacilli. Immune and

even normal serums may at times agglutinate the anthrax bacillus, but the reaction is inconstant, and the ability of an immune serum to cause agglutination is no index of its protective power. Agglutination is somewhat difficult of determination because of the tendency of the bacillus to grow in the form of chains.]

Modes of Infection in External Anthrax.—Only the external anthrax infections, which are the most frequent of all the forms, are of surgical interest. The lesions characteristic of external anthrax follow infections of wounds or develop in the intact skin, apparently from the hair follicles. Naturally, people who, in their employment, come in contact with animals dead of the disease or with the excreta of sick animals, develop this disease most frequently. The uncovered parts of the body are usually attacked. In W. Koch's statistics, comprising 1,077 cases, the head and face were involved 490 times, the upper extremities, especially the hands, 370 times. Apparently the infection is frequently transferred by the hands to the face and other parts of the body, where insignificant wounds, scratches, rhagades, and excoriations provide the infection atria. It is certain that infection follows the use of hides of animals dead of anthrax (caps, pelts, sandals, etc.). The infection may be transmitted by the bites of fleas, or at least transferred by infected fingers when bites are scratched.

Internal Anthrax.—*Pulmonary Anthrax.*—Infections of the lung may follow the inhalation of dust containing spores. Pulmonary anthrax, which is most common in workers in paper factories, who handle and assort rags, appears usually as a double pneumonia and pleurisy and runs an acute course with symptoms of severe general infection, ending fatally in a few days (woolsorter's disease).

Intestinal Anthrax.—The second form of internal anthrax, the intestinal, is rarer than the pulmonary. In this form hæmorrhagic foci, which later become gangrenous, develop, mostly in the intestines. Intestinal anthrax may follow the use of infected food (milk, flesh, and viscera of diseased animals) or the contact of infected fingers with the mucous membranes of the mouth. The symptoms are severe, bloody diarrhœa, peritonitis, and collapse. A general infection rapidly develops and death occurs.

Both of these forms of internal anthrax may accompany an external infection, the two forms developing simultaneously or one being secondary to the other, the infection being carried by emboli (W. Koch).

External Anthrax.—Clinically there are two forms of external anthrax—the carbuncle and the œdema. They develop most frequently in the skin, occasionally in the mucous membranes of the nose and mouth cavity.

Anthrax Carbuncle.—The anthrax carbuncle develops in the beginning from a small reddened, itching area, in which there forms within one, two, or more days a small bluish red vesicle filled with a serohæmorrhagic exudate (anthrax pustule, malignant pustule). There develops very soon, especially if the vesicle is pinched or scratched, a discolored crust, which appears as if sunken in the inflammatory infiltration surrounding it. The gangrenous crust may become as large as a quarter of a dollar or even larger (Fig. 138). Small vesicles with

FIG. 138.—ANTHRAX CARBUNCLE.

serohæmorrhagic contents, from which dark crusts develop, form upon the inflamed, œdematous area. Frequently anthrax bacilli may be demonstrated microscopically and culturally in the exudate which seeps out from beneath the crust, or by injection into mice. If the contents of the vesicles are purulent in character, a secondary infection with pyogenic bacteria has occurred.

Anthrax Œdema.—Anthrax œdema most often accompanies anthrax carbuncle of the face. The cheek and neck may be involved when a pustule develops about the mouth, as the œdema tends to spread rapidly and to involve large areas. It is not sharply defined against the healthy skin as the pustule is. Frequently the skin covering the œdematous and swollen area is markedly reddened, and when this occurs the lesion has been spoken of as anthrax erysipelas. Gangrenous crusts develop from the blebs forming in the inflamed area, and large areas of skin become necrotic.

Lymphangitis and Lymphadenitis.—The lymphatic vessels and nodes soon become involved in both forms of external anthrax and appear as painful swollen cords and nodules. Frequently the lymphatics retain and destroy the bacteria and prevent the extension of the inflammation. If virulent bacteria pass through the lymphatics or pass directly into the blood from fresh wounds, they reach and are deposited in the viscera, especially the spleen and liver, where they develop rapidly and in great numbers. They have been found in the severest fatal cases in the blood, and it is certain that they enter the blood stream

during the course of an infection, but it is much more difficult to demonstrate them than the ordinary pyogenic bacteria in the circulating blood. It is doubtful, however, whether the bacilli multiply in the blood of man as they do in the blood of animals. They may pass through the placenta and the fœtus may become infected.

After death, not only anthrax bacilli, but also different forms of pyogenic bacteria have been cultivated from the blood. The anthrax pustule provides the infection atrium for these secondary infections, which may produce local suppuration and abscesses along the lymphatic vessels and in the lymph nodes.

Fever.—According to K. Müller, local external anthrax is accompanied by fever in only twenty-five per cent of the cases. The fever may become quite high, but as a rule it falls either rapidly or gradually after a few days, if the infection is properly treated. In the severe cases with general infection, characterized by diarrhœa, delirium, and stupor, the fever persists until death, which occurs within a week. Abscesses along the lymphatic vessels and suppuration of the lymph nodes may follow secondary infection with pyogenic bacteria. These secondary infections follow some injury of the pustule, such as scratching or separating the crust. Bacteria then gain access to the lymphatic vessels and a rise in temperature follows this new infection. A general anthrax infection without symptoms of a primary localization (skin, lung, or intestines) is extremely rare.

Diagnosis.—The diagnosis of the external forms of anthrax is not difficult, as the appearance of the local lesions is very characteristic. The diagnosis is made certain by finding the bacilli in the secretion of the bleb, and in that discharged from beneath the crust; by inoculation of mice and cultural tests. The bacilli, as a rule, are found only during the first week of the disease, as they are later destroyed by the tissue fluids and the antagonistic pyogenic bacteria. In the pneumonic and intestinal forms the bacilli may be found in the sputum and fæces.

Prognosis.—The prognosis varies, depending upon the position of the primary pustule or œdema. Anthrax of the head, face, and neck is the most dangerous, and from twenty-three to twenty-six per cent of these cases terminate fatally. Aspiration pneumonia and œdema of the glottis may easily follow the swelling of the neck. According to Nassarow's statistics, the mortality in anthrax of the upper extremity is fourteen per cent, of the lower extremity five per cent. The mortality in internal anthrax is of course much higher (fifty to eighty per cent).

It should be remembered that virulent bacilli may be absorbed and a general infection develop from external anthrax, notwithstanding the fact that only one fifth of the cases end fatally and that the disease remains localized much more frequently in man than in animals. As

a rule the bacilli are destroyed in a short time by the bactericidal substances in the tissue fluids.

Treatment.—In the treatment of external anthrax everything should be avoided which favors the absorption of bacteria. This is most apt to follow scratching, incision and excision of the carbuncle and infected lymph nodes, but may occur after any manipulation, such as cauterization, the frequent injection of antiseptic solutions, the continuous application and changing of moist dressings. Even extensive carbuncles and œdema heal spontaneously, and any such procedures are absolutely unnecessary and often are to blame for the poor results which follow in these cases. In the severest cases, in which a general infection rapidly develops, an operation is no longer of any value.

The chief indication, therefore, is not to injure the infected tissues, as any interference may be followed by an infection of the blood. It is sufficient to cover the inflamed area with a layer of gauze, thickly covered with salve, to prevent rubbing by the bandages. An immobilizing dressing should then be applied and the extremity elevated or suspended. The inflammation and the fever subside under this treatment, and the crust becomes loosened spontaneously during the second week.

It is a mistake to attempt to loosen the crust with tissue forceps, for the granulation tissue is injured in this way and infection atria are provided for pyogenic bacteria. Lymphangitis and abscesses may then develop. The immobilizing dressing should be allowed to remain until the swelling of the lymph nodes subsides and should not be changed too frequently.

Abscesses caused by secondary infections should be incised. Defects of the lips and eyelids resulting from necrosis of the skin should be repaired by plastic operation after the disease has subsided.

How important rest of the infected tissue is in prevention of a general infection may be illustrated by the results of two different experiments made upon mice, which are very susceptible to the disease. Friedrich amputated the tail of a mouse and placed the stump in a bouillon culture of virulent bacilli. He so fixed the animal that the stump would be suspended for some hours in the culture and still not be exposed to any mechanical irritation. Absorption with general infection did not follow, and the animal survived. In the other experiment Schimmelbusch amputated the tail and then rubbed a few drops of a virulent culture into the wound with a knife, and a general infection developed immediately.

Prophylaxis.—In the prophylaxis, the transmission of the disease by infected animals should be prevented. Dead animals, together with their hides, should be buried in deep pits or burned in furnaces. No

part of the dead animal should be used for commercial purposes. Especial care should be exercised in disinfecting the stables. People exposed to infection should be clean and observe the usual precautions which are taken against infectious diseases.

The serum treatment may be tried in the severer cases (*vide* pp. 356 and 357).

LITERATURE.—*Burow.* Ueber die Bekämpfung des Milzbrandes nach der Methode Sobernheim. Berlin. tierärztl. Wochenschr., 1903, No. 35.—*Conradi.* Zur Frage der Toxinbildung bei den Milzbrandbakterien. Zeitschr. f. Hygiene, Bd. 31, 1899, p. 287.—*Friedrich.* Bedeutung des innergeweblichen Druckes für das Zustandekommen der Wundinfektion. Arch. f. klin. Chir., Bd. 59, 1889, p. 458.—*W. Koch.* Milzbrand und Rauschbrand. Deutsche Chir., 1886.—*Lubarsch.* Milzbrand bei Menschen und Tieren. Ergebn. d. path. Anat. von Lubarsch und Ostertag 5. Jahrg., 1898.—*K. Müller.* Der äussere Milzbrand des Menschen. Deutsche med. Wochenschrift, 1894, p. 515.—*Nicolaier.* Zoonosen. Im Handb. d. prakt. Med. von Ebstein und Schwalbe.—*Sobernheim.* Experim. Untersuchungen zur Frage der aktiven und passiven Milzbrandimmunität. Zeitschr. f. Hygiene, Bd. 25, 1897, p. 301; Weitere Mitteilungen über aktive und passive Milzbrandimmunität. Berl. klin. Wochenschr., 1899, p. 273; Milzbrand. In Kolle-Wassermann's Handb. d. pathog. Mikroorganismen. Bd. 2, 1903, p. 1, and Immunität bei Milzbrand. Ibid., Bd. 4, 1904, p. 793.

CHAPTER VI

GLANDERS

Glanders Bacilli.—The bacilli of glanders (Löffler and Schütz) are slender, small, non-motile rods which do not form spores. They stain best with alkaline, aniline dyes. They do not stain by Gram's method; this is important in making a diagnosis. It is rather difficult to stain the bacilli in tissues. Sections should be stained in an alkaline methylene-blue or borax methylene-blue solution, and then destained for some minutes in the solution recommended by Löffler (10 c.c. distilled water, 2 drops of concentrated sulphuric acid, 1 drop of a five per cent solution of oxalic acid).

Culture Media and Glanders in Animals.—The bacilli grow best upon glycerin-agar, but also grow well upon blood serum. Their growth upon gelatin is extremely slow. The virulence of the bacilli diminishes rapidly upon culture media, but may be considerably increased by passing them through animals. Guinea pigs are most susceptible, and die within a few weeks after subcutaneous injections of virulent bacilli. An ulcer develops at the point of injection and the adjacent lymphatic nodes suppurate. Inflammatory and suppurating foci develop in the body, glanders nodes in the viscera, particularly in the lungs, spleen,

and testicles. Suppurative arthritis and ulcers of the nasal mucous membrane complete the clinical picture.

Man appears not to be very susceptible to the disease. In the cases in man, which are not frequent, the infection is transferred in the mucus from the mouth or nose or in the pus discharged from ulcers of diseased animals. Endemics of glanders occur in horses, donkeys, and mules. The infection develops most frequently in the nasal mucous membrane, where nodules, not sharply defined against the healthy surrounding tissue, develop. These nodules are due to cellular infiltration, and soon break down to form ulcers. Large quantities of mucus, which is as infectious as the pus discharged from the ulcers in glanders of the skin, are secreted by the diseased mucous membranes. The disease develops, excepting the rare cases of laboratory infection, almost only in people who come in direct contact with diseased animals.

Infection Atria.—Frequently small wounds of the skin of the hands and face provide the infection atria. Babes and Cornil have demonstrated, however, that bacilli when rubbed into uninjured skin penetrate the hair follicles and may cause a general infection. More rarely in man are the conjunctiva and the mucous membranes of the lip and nose, which are frequently the seat of rhagades and small wounds, primarily infected. Infection of the genital organs and transmission by coitus have been known (Strube).

Character of Local Changes.—Small cellular nodules, which later become necrotic and form ulcers, develop in the mucous membrane when infected. The infection may extend and involve the lower parts of the respiratory tract. If the skin is infected, small cutaneous and subcutaneous nodular infiltrations, which resemble a carbuncle, develop. These are associated with the symptoms of acute inflammation, the epidermis is raised by an exudate and a glanders pustule forms or the area becomes gangrenous, and phagedenic ulcers with undermined edges and dirty floors develop. If the nodules of granulation tissue, which are often accompanied by an erysipelatous inflammation of the skin, extend into the subcutaneous tissue, abscesses develop from which is discharged a thin, foul-smelling pus.

Clinical Forms.—*Acute Glanders—Metastatic Foci.*—These local changes are accompanied by a fever, which not infrequently begins without a chill and general symptoms. The adjacent lymphatic nodes become painful and swollen and suppurate (glanders bubo), and nodules form along the course of the lymphatic vessels from which ulcers and abscesses develop. The bacilli of glanders are carried to different parts of the body by the blood, in which they may be found during life. Many metastatic foci develop in the form of inflammatory nodules, nodular infiltrations, and abscesses. These metastatic foci are produced

by infected emboli, for often many thrombi, loaded with bacilli, are found in the veins of the primary focus. In rare cases suppurative arthritis and osteomyelitis (osteomyelitis malleosa, Virchow) develop. Frequently metastatic abscesses develop in the subcutaneous tissues, and especially in the muscles, while in the viscera, especially in the lung, nodules composed of round cells, which later suppurate and give rise to the symptoms of bronchitis and pneumonia, form. Pustules resembling those of pemphigus and smallpox develop in the skin secondary to the lodgment of bacterial and infected emboli; ulcers likewise develop in the mucous membranes of the respiratory passages, nose, pharynx, and larynx. The skin covering the abscesses and infiltrated areas is reddened. The borders of this redness are sharply defined as in erysipelas, but do not extend. The general symptoms are fever, delirium, coma, vomiting, and diarrhœa. These, in acute glanders, grow progressively worse and persist until death, which occurs in two or three weeks. Death is due either to a general bacterial infection or exhaustion.

Subacute and Chronic Forms.—The subacute and chronic forms may persist for many months or years. Recovery occurs in about one half of the cases, if the acute form does not develop. The local process gradually extends with but few general symptoms, producing large defects in the skin, the infiltration of the skin occurring in the form of large nodules, wormlike or wreathlike strands (the name "worm" has been applied to the chronic forms), from which gradually develop irregular ulcers, fused with each other. If these ulcers have sharp borders and a reniform shape, due to cicatrization upon one side only, so characteristic of the ulcerating gumma, the disease may be mistaken for syphilis, especially if the ulcers are situated upon the lips, the palate, at the entrance to or upon the mucous membrane of the nose. In the chronic form of glanders, metastatic suppurating foci develop only exceptionally, and are then single and occur after long, irregular intervals.

Diagnosis.—The diagnosis of glanders is most difficult in the chronic forms. The acute forms, especially in the beginning or in cases without a demonstrable primary focus, may be confused with typhoid fever, articular rheumatism, or general pyogenic infections. Often the inefficiency of the mercury or potassium iodid treatment of certain lesions of the skin first suggests glanders. It may be mistaken for actinomycosis or tuberculosis, and for that reason the name lupus malleosus has been applied to that form of skin glanders which resembles lupus.

Intraperitoneal injections of pus from a suspected focus into a guinea pig may be employed as suggested by Straus in order to make a positive diagnosis. In two or three days after intraperitoneal injec-

tions of glanders bacilli the testicles become infiltrated and swollen, and a suppurative inflammation of the tunica vaginalis develops, if the animal does not die of a mixed infection. Of course cultures should be made and smears examined in doubtful cases.

Mallein for Diagnostic Purposes.—Mallein, a sterile cultural extract (Kalning, Preusse), is of doubtful value for diagnostic purposes. [" Although it causes a rise in temperature in normal animals when given in considerable doses, the reaction produced in infected animals is so much more intense, and occurs with so much smaller doses that it is generally considered as specific in nature. Some doubt, however, has been thrown on the specificity of the reaction from the facts reported by various observers that toxic substances from other organisms, as tuberculin and preparations from the pneumobacillus of Friedländer, the bacillus pyocyaneus, etc., cause similar phenomena in animals suffering from glanders. Wladimiroff asserts, however, that the reaction caused by these substances differs from that of mallein."—Ricketts, " Infection, Immunity, and Serum Therapy," p. 457.] Bonome is the only one who has reported a febrile reaction after an injection of mallein into a patient sick with chronic glanders. Zieler obtained in two cases neither a general nor a local reaction worth mentioning. Apparently mallein has not the diagnostic significance in man that it has in horses.

Treatment.—As a rule, treatment is powerless in the severe acute forms of glanders. In these cases there is always a general infection, which cannot be prevented by either sparing or destroying the primary focus. Amputation may prevent the general infection, if performed as soon as the diagnosis is made. In the mild forms improvement follows excision of the accessible ulcers and nodules and incision of the abscesses. Sometimes the disease subsides under this treatment and the patient recovers. But all measures, such as curetting with a sharp spoon and rubbing the lesions with gauze saturated with antiseptic solution, which favor the extension of the bacteria to the lymphatic vessels and blood vessels, should be avoided. The resistance and nutrition of the body should be increased and the heart stimulated. Different indications should be met as they arise.

According to Golds, inunctions of mercury ointment act favorably. He tried this treatment in two severe cases which recovered. Other authors have not seen any improvement, even in the mild chronic cases, and the inefficiency of the mercury treatment has led many to change from a diagnosis of syphilis to a diagnosis of chronic glanders.

LITERATURE.—*Bollinger*, in v. Ziemssen's Handb. d. spez. Path. u. Ther., Bd. 3.—*Bonome*. La Riforma med., 1894 (Malleinwirkung).—*Buschke*. Ueber chronischen Rotz der menschlichen Haut. Arch. f. Derm. u. Syph., Bd. 36, 1896, p. 323.—*Ehrich*. Zur Symptomatologie und Pathologie des Rotzes beim Menschen. Beitr. z. klin. Chir.,

Bd. 17, 1896, p. 1.—*Kruse*, in Die Mikroorganismen von Flügge, Bd. 2, 1896.—*Kühne*. Ueber Färbung der Bazillen in Malleusknoten. Fortschritte der Med., 1888.—*Georg Mayer*. Zur Kenntnis des Rotzbazillus und des Rotzknötchens. Centralbl. f. Bakteriol., Bd. 28, 1900, p. 673.—*Preusse*. Berl. tierärztl. Wochenschr., 1898 (Malleinimpfungen).—*Strube*. Ueber Rotzkrankheit beim Menschen. Arch. f. klin. Chir., Bd. 61, 1900, p. 376.—*Virchow*. Die krankhaften Geschwülste. Rotz und Wurm, Bd. 2, p. 543.—*Wladimiroff*. Rotz. In Kolle-Wassermann's Handb. der pathog. Mikroorganismen, Bd. 2, 1903, p. 707;—Immunität bei Rotz. Ibid., Bd. 4, 1904, p. 1020.—*Zieler*. Ueber chron. Rotz beim Menschen, nebst Bemerkungen über seine Diagnose u. medizinalpolizeiliche Bedeutung, den Wert des Malleins. Zeitschr. f. Hygiene, Bd. 45, 1903, p. 309.

CHAPTER VII

ACTINOMYCOSIS

THE ray fungus was first seen by von Langenbeck (1845) in the granular pus of a gravitation abscess, secondary to caries of the vertebræ. Its microscopic appearance was later described by James Israel (1878). A year before this, Bollinger had found a similar fungus in the granulation tumors occurring upon the jaws of cattle. Ponfick demonstrated that the disease in man and animals was produced by the same fungus. O. Israel in 1884 was the first to obtain pure cultures.

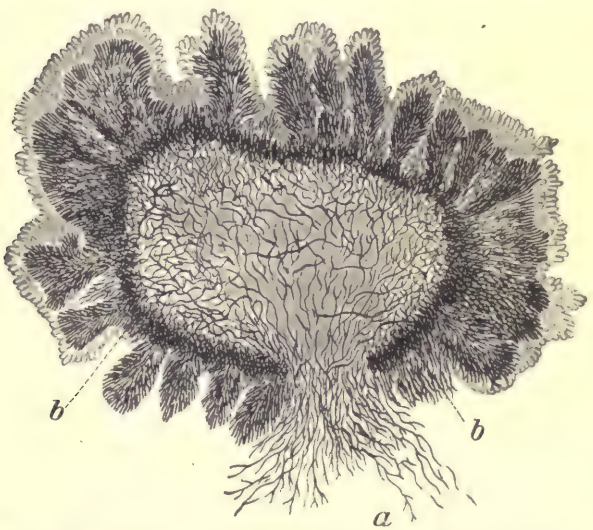

FIG. 139*a*.—SECTION THROUGH A FULLY DEVELOPED COLONY. (After Boström.) *a*, Point at which central filamentous mass breaks through the external layer of clubs; *b*, germinal layer surrounded by clubs.

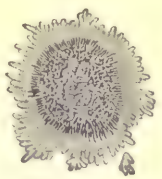

FIG. 139*b*. — SECTION THROUGH A DEGENERATED COLONY. (After Boström.)

The Ray Fungus.—*Morphology.*—There are found in actinomycotic tumors or in the pus discharged from them light yellow granules, vary-

ing in size from a grain of sand to the head of a pin, rarely larger. Each granule represents a colony of micro-organisms. According to Boström, the colony consists of an external layer of radially arranged clubs, the central ends of which become continuous with central filamentous masses (mycelia). There are two zones within the central mass. In the peripheral zone (germinal layer) the filaments are arranged in a radiating manner and run outward in a wavy or spiral course. In the central zone, which is less dense, the fibers interlace and break through the surrounding layer of clubs at one point and grow into the tissues.

The isolated filaments are branched and have a wavy outline, differing in these ways from the ordinary bacteria. Bacilluslike cells and coccuslike bodies may develop from the solid filament. These may become free and, according to Boström, are to be regarded as spores. Entire colonies may develop from single elements of the filamentous mass, from the spores as well as from the fragments of filaments, but not from the clubs. The filaments grow rapidly and produce the disease,

FIG. 140.—ACTINOMYCES. (After Schlegel.) (1) Branched filament with terminal enlargements; (2) long branched filament broken up into bacilluslike structures of different lengths, which are held together by the sheath of the fungus; (3) the division of the filament is still more advanced, indicating the transition of the bacilluslike structure into bodies resembling cocci.

while the clubs are to be regarded as degeneration forms, incapable of further development. Swelling of the end of the filament is the first indication of the formation of clubs. [According to Wright, the radially arranged clubs, which give to the organism the name of "ray fungus," are a manifestation of parasitic existence.] They are found in the

deeper parts of cultures, but are not found in young colonies. In old colonies they form a very thick layer or are calcified.

Growth upon Different Culture Media.—The ray fungus grows upon all media, but somewhat slowly. According to Boström, failures are frequent when old colonies of contaminated material are used, and a number of tubes should therefore be prepared. The growth becomes visible during the first day in the form of small dewdroplike points from which develop, within two weeks, small yellowish red granules. Granules form in bouillon without clouding the media. Many varieties of the fungus, such as those studied by Boström, grow best under aërobic conditions, while others, such as those investigated by J. Israel and Wolff, grow best under anaërobic conditions.

Methods of Staining.—The filaments stain with aniline dyes and by Gram's method. A colony may be doubly stained, using gentian violet for the filaments and picrocarmine and eosin for the clubs. In staining sections, Gram's method may be combined with carmine or the clubs may be heavily stained with eosin and the tissues with hæmotoxylin.

Experimental Inoculation and Botanical Classification.—It is difficult to transmit the disease to animals by inoculating them even with pure cultures. J. Israel and M. Wolf produced, by making intraperitoneal injections of cultures into rabbits and guinea pigs, small granulation tumors which contained the fungus.

There is a wide difference of opinion among authorities as to the exact botanical classification of the fungus. [" By some investigators ray fungi are considered as an independent family midway between the hyphomycetes and the schizomycetes (bacteria) ; others place them under the hyphomycetes in the group of the streptothrix; while still others consider them as pleomorphous bacteria, placing them in the group cladothrix. Petruschky recognizes actinomyces, streptothrix, cladothrix, and leptothrix as genera in the family trichomyces, the latter belonging to the order hyphomyces. Biological variations which have been encountered have led to the recognition of several species of actinomyces, among which are a number of non-pathogenic forms. Wright limits the term actinomyces to those strains which produce colonies of club-shaped organisms in animal tissues."—Ricketts, " Infection, Immunity, and Serum Therapy," pp. 459 and 460.]

Occurrence and Distribution of the Fungus.—The ray fungus occurs upon grains and straw. Parts of grain and parts of straw to which the ray fungus was adherent have been found in the inflammatory swellings produced by the fungus in animals and man. Berestnew could demonstrate fungi, after careful search, upon dried plants, hay, straw, and grains. Liebmann has shown that after inoculating earth with ray fungi, the latter may be found in different parts of planted and

FIG. 141.—A PIECE OF GRAIN COVERED WITH COLONIES OF RAY FUNGI REMOVED FROM AN ACTINOMYCOTIC SWELLING IN THE TONGUE OF AN OX. (After Schlegel.)

germinating vegetables and grains (beans, rye, barley).

Modes of Infection.—The infection in man as well as in animals is transmitted most frequently, as Boström has demonstrated, upon particles of grain. These penetrate the skin, or, in people who are accustomed to chew grain or who accidentally swallow it, pass into the mucous membrane of the mouth cavity, pharynx, œsophagus, respiratory tract, and intestine.

The infection may be transmitted by other foreign bodies (e. g., splinters of wood) to which fungi are attached.

Where the characteristic inflammation is not at first superficial, but develops in the deeper tissues, it is probable that some foreign body has carried the fungus. Grains provided with barbs (*vide* Fig. 141) are apparently able to penetrate deeply into the tissues.

The ray fungus may also pass directly into the tissues. Clinical findings indicate that actinomycosis of the jaw and cheek frequently develops from carious teeth (Israel, Partsch). Partsch has found ray fungi in the cavities of carious teeth, and in one case the fungus had passed down to the end of the root canal. There is no doubt that infection may occur in this way. Other cases in which the symptoms of actinomycosis develop, after a fracture of the mandible, after acute periostitis following extraction of teeth, indicate that the wound or inflammatory focus was secondarily infected with the fungus. In these cases the micro-organisms had apparently been saprophytic for some time.

Action of the Ray Fungus and Character of the Lesions.—The ray fungus produces in the tissues a chronic, progressive inflammation. Its colonies are surrounded by a wide area of granulation and connective tissues,

which are undermined by the fungi. Not infrequently in man it produces tumorlike growths, which in cattle were considered for a long time to be of a sarcomatous nature. The proliferation of the cellular elements is far greater than the exudation and degeneration, which are most marked in other forms of inflammation. The inflammatory new growth is, as von Esmarch has aptly described it, of board-like hardness, and is sharply delimited from the surrounding healthy tissue. It is adherent to the deeper structures and fused with the skin, if the inflammatory process has already extended to the surface. The granulation tissue undergoes fatty changes and becomes liquefied. Small suppurating foci, the skin covering which becomes bluish red in color, develop as the process extends to the surface. Finally these rupture through the skin, and fistulæ are formed from which is discharged pus, which contains the characteristic sulphur-yellow granules (colonies of ray fungi) and necrotic granulation tissue. These fistulæ are chronic and become longer and more branched as new foci develop, which discharge into them. An acute process accompanied by fever and associated with the formation of phlegmons and abscesses is never caused by ray fungi alone. In such cases there is a secondary infection with pyogenic bacteria. When the foci are large and extensive, the general condition of the patient rapidly deteriorates and a cachexia, which may prove fatal, may develop.

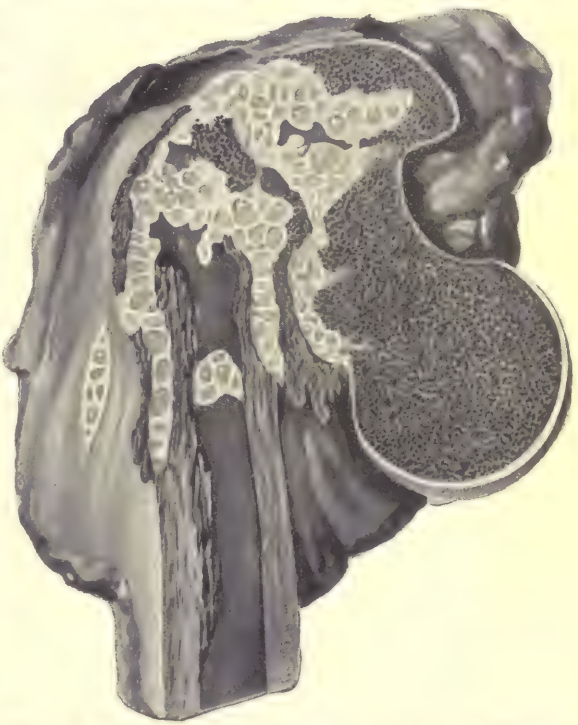

Fig. 142.—Hæmatogenous Osteomyelitis of the Femur Caused by the Ray Fungus.

According to our present knowledge the lymphatic vessels and nodes are not involved in actinomycosis except in rare cases. The infection may be carried by the blood, however, when the ray fungus invades a vein in the primary focus in which a thrombus is developing. Then metastatic foci may develop in any tissue or viscus, even occasionally

in the bones (Fig. 142, Wrede). The clinical course of a general infection with the ray fungus, which is always fatal, is similar to that of the chronic pyogenic infections with metastases.

Actinomycosis in Man.—Depending upon the point at which the infection occurs, actinomycosis in man may be divided into four groups: (1) Actinomycosis of the mouth cavity, (2) of the lungs, (3) of the intestines, (4) of the skin.

Actinomycosis of the Mouth, Face, and Head.—Infections of the face and cheek are placed in the first group. They either develop in the mucous membrane of the cheek directly, or extend from the mucous

FIG. 143.—ACTINOMYCOSIS OF THE FACE AND NECK. (From Bevan's Surgical Clinic.)

membranes of the upper and lower jaws and from carious teeth. In the former case there may be only a slight infiltration of the gum. Anchylosis of the jaw, an early and important symptom of the disease, de-

velops as the inflammation in the cheek extends, and the muscles of mastication become involved in the inflammatory mass. The muscles of mastication are not involved in the small, rapidly softening foci which form about the opening of Stenson's duct (Schlange) and develop in the middle of the cheek anterior to the masseter muscle. Frequently a cordlike, indurated process, extending beneath the mucous membrane of the cheek to the alveolar process or a carious tooth, may be felt. This indicates the way in which the inflammation has traveled. The inflammatory swelling, firmly connected with the underlying bones, extends from the cheek to the submaxillary and temporal regions. The temporal region is also involved when the inflammation extends upward along the internal surface of the ramus of the mandible. Actinomycosis of the maxilla may extend to the orbital and nasal cavities, or may rupture through the base of the skull and produce a fatal meningitis or encephalitis. The process may extend from the jaw or pharynx to the prevertebral tissues, with secondary destruction of the vertebræ. Gravitation abscesses then pass along the anterior surface of the vertebræ and an abscess may develop in the abdominal cavity, secondary to a focus in the mouth or pharynx. Only rarely do central foci develop in the jaw secondary to a focus in an alveolus.

FIG. 144.—ACTINOMYCOSIS OF THE NECK. (After Illich.)

The point of infection in cervical actinomycosis is most frequently in the pharynx, if the disease has not extended from a focus about the jaw. The tonsils and retropharyngeal tissues are most frequently involved first; occasionally the point of infection is in the mucous membrane of the œsophagus or larynx. The transverse, bluish red, indurated folds in the skin in which small subcutaneous abscesses develop are very characteristic of actinomycosis of the neck (Fig. 144). The formation of chronic fistulæ and the discharge of colonies of ray fungi

make the diagnosis certain. The swelling may become so extensive as to render movements of the neck impossible and to interfere with swallowing and breathing.

Actinomycosis of the tongue, occurring in the form of a nodular infiltration, is rare. This may be easily mistaken for a gumma, and if softened, for the ordinary abscess; occasionally for a carcinomatous induration of the floor of the mouth.

Actinomycosis of the Lungs.—Primary actinomycosis may develop in any part of the lungs; most frequently, however, in the lower lobes. This form generally follows aspiration of the ray fungus from the mucous membranes of the mouth or pharynx. Israel's demonstration of a piece of a tooth in a pneumonic focus is very significant from an etiological viewpoint. Most frequently a broncho-pneumonic focus develops; occasionally a superficial catarrhal inflammation. A wide area of the lung surrounding a focus becomes indurated. There is a tendency for this area to become necrotic and to undergo cicatricial contraction. The peculiarity of actinomycosis of the lung, in the early stages resembling tuberculosis, is its tendency to spread to neighboring tissues and not to remain limited to the lungs. The part of the lung involved undergoes considerable cicatricial contraction. When the inflammation reaches the pleura a serous pleuritis develops or the two leaves of the pleura become united and transformed into a thick cicatricial mass. Suppurating foci, which later rupture externally, develop in this newly formed tissue. A number of fistulæ then discharge upon the surface of the chest, or a hard, tumorlike swelling, which later ruptures through the skin, develops over the ribs. The inflammation may extend to the pericardium, or an abscess may rupture through the diaphragm at its point of attachment to the vertebræ and extend to the abdominal cavity or pelvis. Abscesses of the spleen and liver or peritonitis may be caused in this way. A secondary actinomycosis of the lungs develops when an abdominal form extends to the thorax or when emboli, which may be easily carried into the pulmonary veins from the primary focus, lodge in the lung. Ray fungi may be found in the sputum in all forms of actinomycosis of the lungs. If not found, the disease should not, however, be excluded.

Actinomycosis of the Intestines.—Intestinal actinomycosis begins most frequently in the cæcum, and in the parts of the small and large intestine immediately adjacent to it. Other parts of the gastro-intestinal tract (stomach, small intestine, sigmoid flexure, and rectum) are but rarely primarily involved. The symptoms of intestinal actinomycosis develop slowly and are often obscure. Intestinal actinomycosis often resembles acute appendicitis because of fever, local pain, and the position of the induration. If the process extends through the intes-

tinal wall to surrounding tissues, adhesive peritonitis and large indurated masses, resembling tumors, may develop. The necrotic tissue and abscesses, in which are sometimes found particles of grain, apparently the cause of the infection, may rupture in any direction. They may ulcerate into the intestine, bladder, or rectum. Most frequently, however, these abscesses rupture externally. In the latter case, numerous fistulæ open upon the surface of the abdomen, especially in the region of the umbilicus and the right groin, or in the lumbar and gluteal regions, if the abscesses have developed in the retroperitoneal tissue to which the inflammation frequently extends. Fæcal fistulæ are common. In some cases it is difficult to determine whether or not the indurated mass beneath the skin has developed from an intestinal lesion, for sometimes the deep-lying and small mass about the intestine escapes the palpating finger or has already disappeared. The infection may be carried by the portal vein to the liver. Actinomycosis of the latter organ is observed in extensive abdominal actinomycosis as well as in general infections with metastases. Embolic foci develop in the intestinal wall in general actinomycosis.

Actinomycosis of the Skin.—Actinomycosis of the skin is most commonly secondary to one of the forms mentioned above. Primary actinomycosis of the skin does occur after penetrating wounds by foreign bodies, to which ray fungi are attached. Pieces of grain frequently carry the infection. Lupuslike nodules, nodular infiltrations, abscesses, ulcers, and fistulæ slowly develop about the wound. Deep-lying, infiltrated areas, which gradually surround the bone, may resemble clinically a chronic form of suppurative osteomyelitis. For example, von Bergmann observed a case in which the lower part of the femur was surrounded by a large indurated mass. This developed within five years and followed a kick by a horse in which tissues surrounding the bone had been cut.

Diagnosis.—The diagnosis of actinomycosis is difficult, though possible, so long as the foci have not ruptured externally. The microscopic demonstration of the ray fungus in the pus makes the diagnosis certain. The pus must be examined immediately as the fungi lose their characteristic clubs within a day. There may be no colonies in the secretion, and one may look over a number of sections without finding them. The chronic course of actinomycosis may be altered by mixed infection. Acute phlegmons and abscesses then develop, the true nature of which may be recognized when they are incised or, later, when the fistulæ and hard nodules develop. If the inflammatory new growth is prominent, actinomycosis may be mistaken for sarcoma, gumma, or possibly for the enlargement of bone which follows acute pyogenic infections. Examination of indurated cords, extending to the jaw, help

in making a diagnosis of actinomycosis of the cheek. In intestinal actinomycosis the fungi may be found in the fæces; they may be found in the sputum when the lungs are involved.

Prognosis.—The prognosis varies in the different forms. Schlange has made the important observation that the more superficial foci may heal spontaneously after the discharge of the necrotic tissue laden with fungi. The prognosis is most favorable in actinomycosis of the head and neck. Actinomycosis of the lungs and intestines is frequently fatal. The disease always proves fatal when the inflammation extends to vital organs and into deeper parts, especially along the vertebral column into the pelvis, and when a general infection develops through the blood.

Treatment.—In the treatment, naturally, an attempt should be made to aid healing, which occurs when the fungi are encapsulated or discharged externally. It is not necessary to remove the indurated mass, as is the case in the treatment of tumors. The fistulous tract should be followed, and the small granulating and suppurating foci exposed and removed with a sharp spoon. These incisions must be repeated if large indurated masses remain. Under the open treatment with iodoform gauze, complete healing occurs after weeks or months without the injection of antiseptic solutions so often recommended. Potassium iodid is of value. Actinomycosis of the lung and intestine, when not too extensive, may be cured, if the point at which the abscess ruptures is directly over the primary focus (Schlange, Karewski), and if this focus can be exposed by incision of the soft tissues or resection of ribs and the greater part of the focus removed. [In the Bevan clinic, where a relatively large number of cases have been treated lately, excellent results have followed the use of copper sulphate administered in a one fourth grain pill three times a day, and irrigation of the focus, when possible, with a one per cent solution of copper sulphate.]

Foci which are not accessible should be protected from secondary infection. The skin surrounding the fistulæ should be sterilized and aseptic dressings applied. The general condition of the patient should be improved. The fresh air, outdoor treatment employed in cases of tuberculosis is of great value.

LITERATURE.—*v. Baracz.* Ueber die Actinomykose des Menschen. Arch. f. klin. Chir., Bd. 68, 1902, p. 1050.—*Boström.* Untersuchungen über die Aktinomykose des Menschen. Ziegler's Beitr. z. path. Anatomie, Bd. 9, 1890, p. 1.—*Heinzelmann.* Die Endresultate der Behandlung der Aktinomykose. Beitr. z. klin. Chir., Bd. 39, 1903, p. 526.—*Herz.* Ueber Aktinomykose des Verdauungsapparates. Centralbl. f. d. Grenzgeb. 1900, p. 561.—*Hummel.* Zur Entstehung der Aktinomykose durch eingedrungene Fremdkörper. Beitr. z. klin. Chir., Bd. 13, 1895, p. 534.—*Illich.* Beitrag zur Klinik der Aktinomykose. Wien, 1892.—*J. Israel.* Klinische Beiträge zur Aktinomykose des Menschen. Berlin, 1885;—Neue Beobachtungen auf dem Gebiete der Mykosen des

Menschen. Virchow's Arch., Bd. 74, 1878, p. 15.—*J. Israel* und *M. Wolff.* Ueber Reinkultur des Aktinomyzes und seine Uebertragbarkeit auf Tiere. Virchow's Arch., Bd. 126, 1891, p. 11 and Bd. 151, 1898, p. 471.—*O. Israel.* Ueber die Kultivierbarkeit des Aktinomyzes. Virchow's Arch., Bd. 95, 1884, p. 140.—*Karewski.* Beitrag zur Lehre der Aktinomykose der Lunge und des Thorax. Verhandl. der Berl. med. Ges., 1899.—*Kruse,* in Die Mikroorganismen von Flügge, Bd. 2.—*Lachner-Sandoval.* Ueber Strahlenpilze. Strassburg, Beust, 1898.—*Lieblein.* Ueber die Aktinomykose der Haut. Beitr. z. klin. Chir., Bd. 27, 1900, p. 766;—Ueber die Jodkaliumbehandlung der menschlichen Aktinomykose. Ibid. Bd. 28, 1900, p. 198.—*W. Müller.* Ueber Aktinomykose der Brustkrüse. Münch. med. Wochenschrift, 1894, p. 1027.—*Petruschky.* Die pathogenen Trichomyzeten. Handb. der pathog. Mikroorganismen von Kolle-Wassermann, Bd. 2, 1903, p. 832.—*Poncet* et *Berard.* De l'actinomycose humaine en France. Gaz. hebdom. de méd. et de chir., 1902, No. 27.—*Schlange.* Zur Prognose der Aktinomykose. Arch. f. klin. Chir., Bd. 44, 1892, p. 876.—*Schlegel.* Aktinomykose. Handb. der pathog. Mikroorganismen von Kolle-Wassermann, Bd. 2, 1903, p. 861.—*Silberschmidt.* Ueber Aktinomykose. Zeitschr. f. Hygiene und Infektionskrankh., Bd. 37, 1901, p. 345.—*Tusini.* Ueber die Aktinomykose des Fusses. Arch. f. klin. Chir., Bd. 62, 1900, p. 249.—*Wrede.* Hämatogene Osteomyelitis durch aktinomyzes. Chir.-Kongr. Verhandl., 1906.—*Bevan* and *Post.* Actinomycosis. Chicago Medical Recorder, Oct., 1905.

CHAPTER VIII

MADURA FOOT

By Madura foot is understood a characteristic, chronic, progressive inflammation which occurs most frequently upon the feet; occasionally upon the hands. It was first observed in Madura (India, 1712), then in Hindustan. Occasionally cases are seen in America and Africa.

The disease begins with a painless swelling of the sole of the foot, usually only one foot being involved. It runs a chronic course, during which round, bluish red, confluent, soft nodules form, which rupture, leading to the formation of chronic fistulæ. The latter heal, but soon break open again. Gradually the inflammation extends to the dorsum of the foot and extends deeper, involving the tendons and joints, the periosteum, and bone, the latter being completely destroyed or transformed into a number of cavities. The pus discharged from these lesions is thin, foul-smelling, and contains small yellow or black granules.

The Madura foot fungus, which grows well upon glycerin-agar, apparently belongs to the streptothrix group. [" Pure cultures of the organism, which is called streptothrix maduræ (Vincent) were first obtained by Vincent in 1894, and have been studied by a number of observers since that time. It bears a close resemblance to the actinomyces, and by some is considered a variety of that organism. Differences between the black and yellow varieties are not clearly set forth."

—Ricketts, " Infection, Immunity, and Serum Therapy," p. 462.] The absence of clubs differentiates it from the ray fungus. The tissue changes produced by this fungus are similar to those produced by the ray fungus.

Permanent cure follows amputation. If the part involved is not amputated, the patient gradually loses strength and becomes cachectic. In the beginning of the disease incision and curettage of the separate foci may be tried.

LITERATURE.—*Babes.* Der Madurafuss. In Kolle-Wassermann's Handb. der pathog. Mikroorganismen, Bd. 3, 1903, p. 454, with Lit.—*Bollinger.* Ueber primäre Aktinomykose der Fusswurzelknochen. Münch. med. Wochenschr., 1903, p. 2.

CHAPTER IX

BLASTOMYCOSIS [1]

Introduction.—Blastomycosis was first brought to the notice of the scientific world in May, 1894, since which time its position has been attacked and defended by able men. At present there are only a few who are unwilling to accept the disease as an entity. During the past four years the importance of the affection has been emphasized by multiplication of the number of cases recognized (now more than one hundred) and by the gravity of the disorder when general infection occurs. Fifteen or more systemic cases have been recognized and studied. Among other names prominently connected with the development and study of the disease clinically, experimentally, microscopically, etc., should be mentioned Gilchrist, Busse, Buschke, Hyde, Montgomery (Frank Hugh), Hektoen, Bevan, and Ricketts.

Since general infection has been recognized as a prominent feature of the disease, the term blastomycosis has been generally adopted, the original term, blastomycetic dermatitis, given by Gilchrist, being too narrow. In those cases where the infection is confined to the skin, the term " cutaneous blastomycosis " is applied, while the generalized infections are usually designated as " systemic," " generalized," or " disseminated " blastomycosis.

History.—In May, 1894, Gilchrist demonstrated sections of a " peculiar skin disease " before the American Dermatological Association, in which the organisms of this disorder were noted and described. The previous clinical diagnosis in this case, made by Duhring, was " scrofu-

[1] For complete abstracts of cases of systemic blastomycosis, see Transactions of Sixth International Congress of Dermatology, 1907.

loderma." Gilchrist and Stokes, in July, 1896, made a report of this case, and again more fully in 1898. Six months after Gilchrist's demonstration of the sections, Busse published an article reporting his case, and again with Buschke in a more extended study with a report in 1895 and 1899. In 1896 Curtis reported his study of a case of what he termed "saccharomycose humaine." Then followed reports of cases termed "blastomycetic dermatitis," the term adopted by Gilchrist, by the following gentlemen: In 1898 Wells, Hessler, Hyde, Hektoen, and Bevan, with a further study of the organism from the latter case by Hektoen in 1899; in 1899 Owens, Eisendrath and Ready, and Murphy and Hektoen; in 1900 Anthony and Herzog, Coates, Baldwin, Brayton (three cases in April, 1900, July, 1901, and February, 1902); Montgomery (Frank Hugh) (case reported before the American Dermatological Association), with a further report, with two additional cases by Montgomery and Ricketts in January, 1901; in 1901 Dyer, Stelwagon, Harris, etc., since which time cases have multiplied until a large number has been recorded. From early in 1903 until the present time cutaneous blastomycosis has not been reported to any extent from Chicago; not that observations have been wanting, for new cases are constantly coming under observation.

Notwithstanding the fact that in the second recorded case of blastomycosis (that of Busse) a general infection occurred, much doubt has existed concerning general infection; this doubt, however, has been dispelled by subsequent observations.

The first recorded general infection was that of Busse in 1894, the second that of Montgomery and Walker in April, 1902. Further reports of cases, with study more or less complete, frequently including autopsy records, have been recorded by Ormsby and Miller in March, 1903; by Cleary in May, 1904; by Eisendrath and Ormsby in October, 1905, with additional clinical findings and autopsy report in the same case by LeCount and Meyer in March, 1907, and Bassoe in December, 1905; in 1906 by Irons and Graham and Hektoen and Christenson (two cases); in 1907, by Baum and Stober (demonstrated before the Chicago Dermatological Society); by Garvy (paper read before branch of Chicago Medical Society) and Montgomery (Frank Hugh), this case was demonstrated by Dr. Montgomery before the Chicago Dermatological Society in April, 1905. In addition to these, we have seen three cases not recorded.

Blastomycetes were first demonstrated in the *sputum* in the case of Eisendrath and Ormsby in 1905, and in *fecal matter* from the same case in 1906 as recorded by LeCount and Meyer.

Geographical Distribution.—In the United States, Chicago is apparently the center of infection, as the majority of cases, both of cutaneous

and systemic blastomycosis, have been recorded there. In addition, cases have been noted in Indiana, Wisconsin, Nebraska, Texas, Massachusetts, Kentucky, Colorado, Utah, Maryland, New York, Michigan, Minnesota, and Iowa. Among the foreign countries may be mentioned Canada, England, Germany, France, Scotland, Japan, India, Italy, and South America.

Organs and Tissues Involved.—In the victims of the disorder coming to the post-mortem table, blastomycetes have been demonstrated in the following organs: larynx, trachea, lungs, pleura, myocardium, liver, spleen, pancreas, kidneys, adrenals, lymph glands, bones, joints, subcutaneous and cutaneous tissues, brain, spinal cord, and colon. The organisms have been demonstrated in sputum and in fecal matter.[1]

FIG. 145.—CUTANEOUS BLASTOMYCOSIS SHOWING DELICATE SCAR TISSUE IN THE CENTER WITH ACTIVE ADVANCING BORDER.

Clinical Symptoms in Cutaneous Cases.—The age of the patient has varied from twelve to seventy-four years. Lesions have occurred over practically the entire cutaneous surface, the face having been the site of election in a great number of cases; the region about the cheeks and eyelids is frequently attacked. The size of the lesions has varied from a small beginning papulo-pustule to large patches several inches in diameter. In the case used for illustration (Figs. 145, 146, and 147) nine patches ex-

[1] Since the above was written, blastomycetes have been found in the prostate gland and in the urine.

isted, some of which covered an area of several inches. The lesion begins as a small papule, or papulo-pustule, which spreads peripherally, eventually forming a patch of varying dimensions. A patch the size of a silver quarter presents the following characteristics:

It is surrounded by a bluish red areola in which the small miliary abscesses characteristic of the disorder are found. The areola gradu-

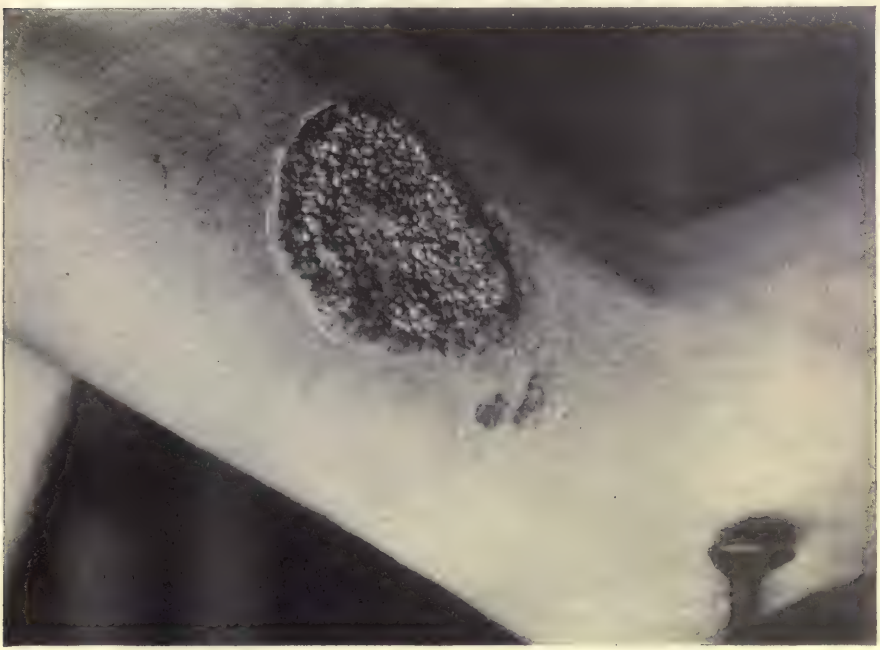

FIG. 146.—CUTANEOUS BLASTOMYCOSIS SHOWING CIRCULAR PATCH WITH PAPILLOMATOUS ELEVATIONS COVERING THE SURFACE.

ally slopes from the elevated patch to the normal surrounding skin, and is about one fourth of an inch in width. The main part of the lesion is elevated about an eighth of an inch, and the top of the patch is more or less flat, papillomatous or verrucous, crust-covered, or discharging, or superficially ulcerated. Pus may be squeezed from between the papillomatous projections, as in verrucous tuberculosis. The miliary abscesses in the sloping border of the patch are characteristic, and vary from minute, scarcely visible points to lesions the size of a pin's head, and from which a glairy, muco-purulent material can be obtained, from which the organisms may be recovered in pure culture. In parts of the patch which have undergone involution a superficial scar is left, which is usually soft and smooth, but may be irregular and corded. The scar, however, is not, as a rule, disfiguring except

when near the eyes, in which case more or less extensive ectropion occurs.

The course of the disease is, as a rule, chronic in the cutaneous cases, periods of activity and spreading alternating with periods of apparent

Fig. 147.—Cutaneous Blastomycosis Showing Patch on Dorsum of Foot.

quiet. The patches may be in close proximity or separated by quite a distance, as, for example, when the patch exists on the face near the eye, with another on the wrist; or again, one patch on the face and another on the leg near the knee, or at the same time on the face, arms, legs, etc., various sized patches and in varying degrees of activity may be found. Often in the scar of an apparently healed area the small abscesses containing the organism are noted, which may light up into activity at any time. As Montgomery states: "A single patch may at one time present all the stages of the disorder, showing at the same time several of the following features: the advancing border, new lesions forming on old scars, verrucous or cauliflower lesions in various stages of development or disappearance, a base in places dry and firm

and in others soft and infiltrated with muco-pus, a scar tissue in part thick and irregular and in part smooth, soft, supple, and non-attached to the deeper tissue."

Cutaneous lesions occurring in systemic cases are described under that heading.

Clinical Symptoms in Generalized Cases.—The observations recorded in this chapter were made from material collected chiefly about Chicago, with reference also to findings recorded in literature from other parts of this country as well as abroad, but no effort has been made to incorporate all the recorded cases, as that would be beyond the scope of this article.

In collecting data for describing the clinical symptoms and pathological findings of systemic or generalized cases, fifteen recorded and

FIG. 148.—CUTANEOUS LESIONS IN A PATIENT THE SUBJECT OF GENERALIZED BLASTOMYCOSIS. (Courtesy of the *Jour. of Cut. Dis.*)

unrecorded cases are considered. In twelve of the patients the disease proved fatal. One has apparently recovered (Garvy) and two others are either well or nearly so at the last report. Three of the cases the writer has studied carefully, and through the courtesy of his colleagues has observed and to some extent investigated six others. Of these nine

only one is living. It is readily seen, therefore, that when systemic involvement occurs it assumes a grave character. Some of the cases have proved rapidly fatal.

The symptoms include those arising from infection of practically all of the organs of the body, but apparently the symptoms presented clinically are not proportionate to the marked findings demonstrated at the post-mortem table. For example, in the case of Ormsby and Miller, only mild physical findings referable to the lungs were noted before death, and the patient had only a moderate cough with scanty expectoration, yet the lungs were completely infiltrated with the tubercles and abscesses peculiar to the disorder. The kidneys, too, were infected, but urinary findings during life were negative.

Some general symptoms, however, have been more or less constant. An *irregular temperature* has been the rule, ranging from 98½° to 103° F. One subject, however, had constantly a subnormal temperature, 96° to 98° F. (Cleary). *Emaciation* has been constant, and in some cases extreme. *Weakness*, with *prostration* and different grades of *anæmia* have occurred. *Albumin* with *casts* showing nephritis has been noted. This was present to a high degree in the case of Cleary. *Cough* with sanguineo-purulent expectoration has been present in several cases in which blastomycetes have been demonstrated (Eisendrath and Ormsby, Bassoe, Irons and Graham, and others). The *pulse* and *respiration* have been proportionate to the temperature in most cases. However, as emaciation increased, a feeble, rapid pulse was the rule. *Pain* occurred in the chest in one case, and *severe pain* in the back in another. Œdema of the extremities, as well as of the face, has been noted. One almost constant accompaniment has been the formation of *multiple subcutaneous and cutaneous abscesses* and nodules, which later have developed into *ulcers*, their distribution at times involving almost the entire body. The ulcers in these cases result from the breaking down of the subcutaneous abscesses, and at times cover large areas by subsequent burrowing and destruction of tissue. The ulcers are irregular in outline, ill-conditioned, discharging, or crust-covered, at times having fistulous connections with deeper structures. Occasionally metastatic abscesses have been sufficiently large to hold within their walls as much as a liter of pus. The pus in all these has been a product of the blastomycetes, as no other germ has been discovered, while these organisms have been obtained in pure culture. *Diarrhœa*, with the organism in the fecal matter, was noted by LeCount and Meyer in the case of Eisendrath and Ormsby. *Spondylitis* has occurred several times. In one case several vertebræ were destroyed with a corresponding amount of spinal cord. In addition to the vertebræ, some of the ribs, the tibia, and the cranial bones have been involved. Suppurative ar-

thritis has occurred in several cases, large quantities of pus having been found in the joints. Blastomycotic laryngitis has developed in two cases, in one of which the organisms were demonstrated in the ulcers in the larynx (Ormsby and Miller). In one case lesions were demonstrated in the brain.

The lymphatic glands have been exceptionally free, but in several cases involvement of these organs has been noted.

The general picture, then, has been that of constitutional involvement, somewhat similar to tuberculosis, for which disorder it has been at times mistaken. Once (Walker and Montgomery) the diagnosis, both clinical and by autopsy, was made of tuberculosis, but on later investigation blastomycetes were demonstrated both in the skin and internal organs, with no tubercle bacilli. Miliary tubercles or nodules of blastomycosis strongly suggest those of tuberculosis.

To sum up, the clinical picture is nearly as follows: Evidence of general infection, exhibited by *irregular temperature, loss of appetite, general weakness, emaciation, cough*, with sanguineo-purulent or possibly only frothy expectoration, *rapid, feeble pulse*, acceleration of the respiration, at times *albumin* in the urine, *multiple subcutaneous nodules and abscesses* resulting in superficial irregular ulcers, abnormal physical findings in the lungs, such as dullness, bronchophony, bronchial breathing, various râles, etc., œdema of the extremities, and various grades of anæmia. A combination of generally distributed, *subcutaneous nodules, abscesses, and cutaneous ulcers, with evidence of constitutional disease, should always suggest generalized blastomycosis.*

Cutaneous Histopathology.—The original description given by Gilchrist in his case, the first one recorded, has had few essential additions during the many years that have since elapsed, and his findings have been corroborated by practically all observers. The resemblance in the histological architecture between many of these cases and some forms of cutaneous tuberculosis is striking. The epidermal hypertrophy, the cellular infiltration in the corium, the partial or complete destruction of collagen and elastin in areas most markedly affected, the presence of many giant-cells, the formation of tubercles or pseudotubercles—all are found in both disorders. The striking and characteristic miliary abscesses in both the epidermis and corium, showing marked evidences of inflammatory action and containing the organisms peculiar to blastomycosis, mark the special difference.

In blastomycosis the chief pathological changes occur in the epidermis and the upper portion of the corium. The stratum Malpighii is hypertrophied, sending prolongations in various directions into the corium. In this layer miliary abscesses of various sizes occur. They contain chiefly polymorpho-nuclear leucocytes, fragments of epithelial

cells in various stages of degeneration, parts of nuclei and other detritus, with one or several of the causative organisms, the latter usually in pairs. The abscesses vary in size from those only sufficiently large to contain a few leucocytes with one organism, to those sufficiently large to be easily seen with the naked eye. The wall of the abscess consists of more or less flattened epithelial cells. Occasionally the abscesses contain in addition giant-cells, and at times a few plasma-cells. The rest of the rete is œdematous, its cells being swollen; the leucocytes are distributed irregularly about and between the cells. Hypertrophy

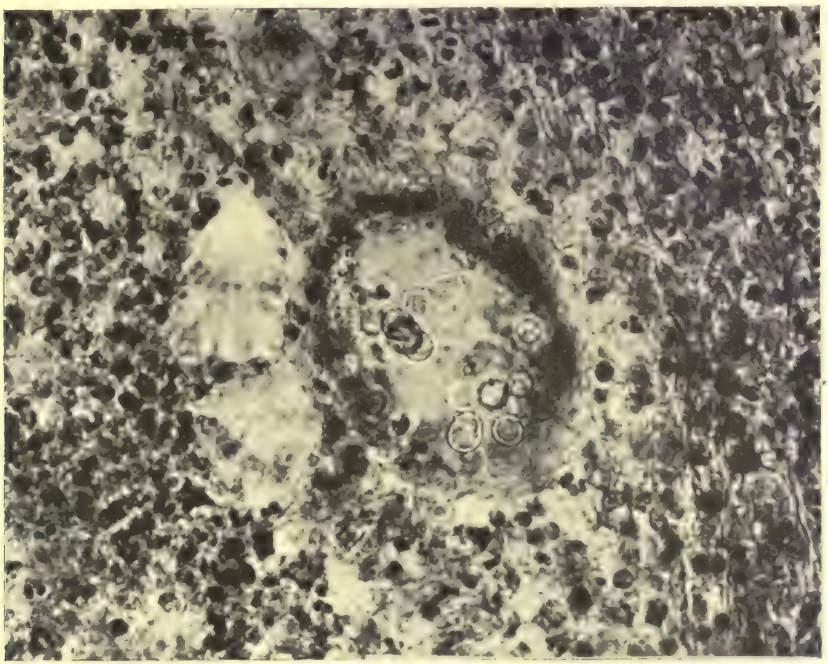

FIG. 149.—MICROPHOTOGRAPH. CUTANEOUS SECTION (HIGH POWER) SHOWING GIANT-CELL CONTAINING ORGANISMS OF BLASTOMYCOSIS. (Courtesy of the *Journal of the American Medical Association.*)

of the rete (acanthosis) is often so marked as to suggest an epitheliomatous change, but with careful study the basal layer is always found intact. The surface of the epidermis is irregular, and is covered with epithelial cell débris, fibrin, pus- and blood-cells.

The corium is the seat of a cellular infiltration. In the more acutely inflamed areas miliary abscesses occur similar to those in the epidermis. Those in the center contain one or several organisms surrounded with leucocytes, which in turn are surrounded by connective tissue and plasma-cells, with many giant-cells interspersed. The giant-cells often

contain the organism singly or in pairs; several may be present. At times a space is noted, from which an organism may have escaped (Ricketts). Mast-cells may be few or abundant, and of various shapes, depending on their evolution. In the areas of dense infiltration collagen and elastin are diminished in amount or absent. The appendages of the skin are not actively involved. In the more active areas miliary abscesses with numerous organisms and leucocytes predominate; in more chronic areas, giant- and plasma-cells are in excess. In some sections large numbers of classical plasma-cells are noted. Hyaline degeneration is described in these cells by Ricketts.

General Pathology.—The findings recorded here, both gross and microscopic, are collected from the eight cases now recorded, with autopsy reports.

In view of these findings, the striking and characteristic changes consist in the presence of nodules or tubercles and abscesses in the soft tissues, and caries in the bones. The organism of blastomycosis is markedly pyogenic. The nodules and abscesses vary greatly in size, location, and number in different cases, but are always present. The characteristic composition of these lesions in all areas consists, primarily, of the organism of blastomycosis in varying numbers, leucocytes (polymorpho-nuclear chiefly, but also mononuclear) and giant-cells; secondarily, of plasma- and mast-cells, cellular and other detritus, pigment, red blood cells, etc., the latter being more or less abundant, depending on the location and acuity of the process. The abscesses vary in size from those which are microscopic to some sufficiently large to hold one half liter of pus. They may occur in all the organs of the body, as well as in the bones and joints, and are especially characteristic in the subcutaneous tissue, where at times fistulæ and large excavations are produced. These abscesses have also been noted in the glands, both abdominal and thoracic, behind the œsophagus and in the bones (vertebræ, ribs, tibia, etc.).

Fig. 150.—Cut Section of the Spleen Showing Areas of Blastomycotic Infiltration. (Photograph courtesy of the *Journal of Cutaneous Diseases.*)

A typical blastomycotic nodule has in its center an area of necrosis, with blastomycetes, leucocytes, and cellular detritus surrounded by giant-cells. It may contain also connective tissue, plasma-, and mast-cells.

In some cases great destruction of tissue has occurred in certain of the internal organs. The lungs in all cases have been the seat of marked changes. The nodules and abscesses may infiltrate almost the entire organ. The presence of large numbers of blastomycetes with giant-cells and leucocytes is characteristic. Giant-cells always contain from one to several organisms. Plasma- and mast-cells, much pigment, and granular detritus are also found, and in some areas newly formed connective tissue. In one case a lung was almost completely destroyed. It contained an enormous number of organisms in various stages of development. The process in most cases is a blastomycotic broncho-pneumonia.

Blastomycotic nodules or tubercles are found in the pleura, in peribronchial lymph glands, and in the myocardium.

In the abdominal cavity the spleen has been the seat of most destructive changes, consisting of nodules, areas of necrosis containing granular detritus, and large numbers of the parasites. Here giant-cells have not been conspicuous. The liver, kidneys, adrenals, pancreas, lymph-glands, and colon have all been the seat of similar changes to a less degree, except in one case where the adrenals (Cleary) were extensively invaded. Like the spleen, the adrenals contained no giant-cells.

The destructive process has been marked in the spinal column in several cases. In one case several vertebræ were destroyed, with a corresponding length of the spinal cord. The necrotic areas in the bones contain the organism in large numbers, leucocytes, an occasional giant-cell, and a fibrinous exudate.

Amyloid degeneration is not constant, but has occurred in several cases and may be extensive. It has been found in the liver, kidneys, adrenals, spleen, retroperitoneal, mesenteric, and mediastinal lymph-glands, and colon.

Summary of Gross Pathological Findings.—Blastomycotic subcutaneous abscesses, nodules, sinuses, ulcers, and scars, covering practically all parts of the body. Blastomycotic laryngitis and broncho-pneumonia. Blastomycosis of the pleura, subpleural and retropharyngeal tissue, the peribronchial lymph-nodes, the liver, spleen, kidneys, adrenals, colon, various bones (tibia, ribs, vertebræ), the external surface of the spinal dura mater, the spinal cord, the cerebellum, various joints (elbow, knee, ankle, etc.), chronic parenchymatous nephritis, atrophy of the heart, etc. In addition to the above, blastomycotic areas have been demonstrated microscopically in the myocardium, pancreas,

and various lymph glands, and amyloid degeneration in the organs previously mentioned.

Description of the Organism in Tissue.—The biological position of this organism has not been positively settled. In tissue its method of reproduction is by gemmation. It is made up of a capsule (at times an adventitious capsule in addition), a clear zone, granules, and at times a vacuole. The size varies from 5 to 15 μ, but we have many times noted organisms as large as 30 μ. Taken as a whole, the organism is round or oval or somewhat irregular, surrounded by a homogeneous, doubly contoured, refractile capsule, immediately within which is a clear zone, while the center contains granules of various sizes and shapes, and sometimes a vacuole. These protoplasmic granules are at times basophilic, demonstrated by their taking the red part of Unna's polychrome methylene blue stain.

FIG. 151.—SMEAR FROM TUBERCLE-LIKE LESION IN THE SPLEEN, MOUNTED IN ONE PER CENT POTASSIUM HYDRATE IN GLYCERIN, SHOWING THE ORGANISM OF BLASTOMYCOSIS (× 1000). (Microphotograph, courtesy of the *Jour. of Cut. Dis.*)

While endogenous spore-formation is not proven in these organisms, one may often see ruptured capsules, and in the immediate neighborhood small granules similar to those within the capsule. At times when numbers of organisms are present, crescentic-shaped capsules partly filled may be seen.

The organism is well seen in fresh pus or tissue mounted in a ten per cent solution of potassium hydrate, and may be easily stained with any of the common aniline dyes.

Cultural Characteristics.—In common with other fungi, blastomycetes present multiform cultural appearances, depending upon the media used and the temperature at which they are grown. Ordinarily they grow well on glycerin- and glucose-agar, blood-serum-agar, and in broth.

As a rule, a moist, pasty growth occurs on glycerin-agar at room temperature, with only moderate aërial hyphæ, while more aërial hyphæ form on glucose-agar and agar-agar at the same temperature. Cultures begin to develop in from two to fourteen days, subcultures usually in from two to five days. Mycelial formation is more abundant during the first few weeks, but later budding-forms occur. In the incubator the growth is more moist and pasty, and budding-forms are more numerous early. These latter facts have recently been again demonstrated by Hamburger. Otis and Evans, studying the growth of the organism isolated from a case of systemic blastomycosis reported by Ormsby and Miller, noted that in thirty hours, after a hanging drop culture was made in bouillon, the cells started processes which grew 60 μ in length during the following twenty-four hours, the mycelia being homogeneous and possessing a very thin cell wall; later protoplasmic granules and pinkish vacuoles appeared. After several days a subdivision of the mycelia into segments of varying lengths occurred. After a hanging drop culture in glycerin-agar had developed for about a month, there appeared an end-cell that budded, and in the course of several days a group of these cells was noted. It may be that the organisms develop in tissue by budding only; on media by segmental mycelial formation with lateral conidia, and later by a certain number of budding forms, the latter differing, however, in some particulars, from the forms seen in fresh tissue and pus.

Fig. 152.—Growth of the Organism of Blastomycosis on Glycerin-agar Twenty-one Days Old, from a Miliary Abscess in the Spleen, Showing Moist, Pasty, and Wrinkled Growth. (Photograph, courtesy of the *Jour. of Cut. Dis.*)

Animal Experiments.—Both local and general infections have been produced experimentally in mice and guinea pigs. The latter are

rather resistant to infection, but a general infection may be produced and the organism later recovered from practically all the viscera when large doses of the organism in culture are injected. The lungs, liver, spleen, kidneys, testicles, diaphragm, etc., have been the seat of tubercles peculiar to the disorder in which the causative organism has been demonstrated and recovered in pure culture. The histological picture presented in these various organs has been similar to that found in

FIG. 153.—SMEAR FROM GROWTH OF THE ORGANISM ON MEDIA FIVE WEEKS OLD MOUNTED IN A ONE PER CENT POTASSIUM HYDRATE SOLUTION. (Microphotograph, courtesy of the *Journal of the American Medical Association.*)

the human body. The testicles have usually been infected, and in many cases large, caseous masses in which the budding organisms occur abundantly have developed. After the injection of moderate doses, guinea pigs have been ill for a time, and have then recovered. The best results for the purposes of demonstrating pathological findings with the organism have been obtained by giving large doses and then killing the animal about three weeks afterward.

Infection Atrium.—In some of the cutaneous cases a history of preceding trauma could be obtained. This apparently opened the way for infection. In the majority of the systemic cases, the lungs have been accepted as the point of origin of the infection.

It is an interesting fact that during the post-mortem examination of the case reported by Ormsby and Miller, one of the attendants was infected with, and later developed blastomycosis, this case being, without question, one of direct inoculation. In only one instance is there a record of more than one case occurring in a family. Three members are reported with the disorder by Eastman and Keene.

Comparison between Blastomycosis and Coccidioidal Disease.—A group of cases have been described under the title of protozoic disease, protozoic dermatitis, dermatitis coccidioides, and granuloma coccidioides.

The first was recorded by Wernicke, of Buenos Ayres, as a doubtful mycosis fungoides. The second and third cases were studied and reported by Rixford and Gilchrist. Others have been recorded by D. W. Montgomery, Rykfogel, Morrow, Ophüls, and Moffett. In a recent report (October, 1905), with a review of the cases, Ophüls collates ten recorded and adds three new cases, making a total of thirteen.

This disease resembles very closely blastomycosis, both clinically and microscopically. The protozoic disease is caused by an organism that resembles in many particulars the germ of blastomycosis. The organism found in the California cases is large, circular, double-contoured, and develops in tissues by endogenous spore-formation. It varies in size from 15 to 30 μ in diameter and contains sporules, sometimes as many as a hundred in a single capsule, which escape upon rupture of the capsule and develop into adult forms.

Prickles or long spines are described covering the capsule. On media the organism grows as a mold fungus. Like blastomycosis, it produces lesions in skin and in internal organs resembling tuberculosis. The cutaneous lesions are granulomatous and may appear as papulopustules, ulcers, tumors, etc. Cutaneous lesions may precede the general infection, or be secondary, as in blastomycosis. The cases, as a rule, have been fatal. The majority of the cases have been reported from California. Since a more extended study has been made of the generalized cases of blastomycosis, the early differences thought to exist between the two disorders have been reconciled. At present the only constant difference is found in the mode of reproduction of the two organisms in tissue, the one by budding, the other by endogenous spore-formation. On media, too, many differences may be pointed out, but these differences are no greater than those observed between different cultures of undoubted blastomycetes. It is maintained by D. W. Mont-

gomery that potassium iodid has little or no effect either in the cure of or inhibition of the progress of coccidioidal disease, while there is no question as to its value in blastomycosis. It seems, therefore, that with so many points of resemblance, and with so few differences, the two diseases should be classed as members of the same group. It is justifiable to assume that climatic differences play a part. One patient suffering from generalized blastomycosis made a complete recovery on going to California, but large doses of potassium iodid were administered for a long period before leaving Chicago (Garvy).

Diagnosis.—Tuberculosis is the disease which is most apt to be confused with systemic blastomycosis. In one case, recorded several years ago, the diagnosis of tuberculosis was made both clinically and at autopsy. It has been the aim during the developmental period of the studies of the disease to positively exclude the presence of the bacillus of tuberculosis. In two cases, in the study of which the writer was interested, tuberculin injections were made with negative results. Guinea pigs were inoculated with pus from cutaneous and subcutaneous lesions, and also with tissue and tubercles from internal organs, always with negative results as far as tubercle bacilli were concerned. Large numbers of smears from various areas, as well as sections of tissue from the same areas, were examined for tubercle bacilli with negative results. The only parasite found microscopically, experimentally, and culturally was the organism of blastomycosis.

In any case of doubtful general tuberculosis the sputum, as well as pus from the cutaneous or subcutaneous lesions, should be examined for blastomycetes.

The cutaneous cases have been mistaken most often for verrucous tuberculosis, less often for syphilis, and occasionally for epithelioma.

The chief points of difference between blastomycosis and verrucous tuberculosis are: In blastomycosis the edge of the patch is more inflammatory and contains the characteristic miliary abscesses, from which the organisms can be readily removed, examined in a ten per cent solution of potassium hydrate, and be readily seen. There are apt to be several patches, and these develop with great rapidity. In verrucous tuberculosis the site of election is often the dorsum of the hand alone, while in blastomycosis, if this region be involved, other areas also, such as the face near the eyelid, are likely to be affected. In case of doubt a histological section reveals the true nature of the process. Miliary abscesses in the epidermis and the corium, containing leucocytes and blastomycetes, make the diagnosis positive. Cultural experiments also may be made. The smooth, supple scar of blastomycosis is characteristic.

The late lesions of syphilis are the only ones that could possibly be

confused with the lesions of blastomycosis. The circinate lesions of lues, made up of individual tubercles and characteristic ulcer- and scar-formation, differ from the regular, circular, or oval patches of blastomycosis with characteristic edges, miliary abscesses, etc. The latter may persist for long periods in the same area, while a lesion of syphilis is apt to heal and advance to new areas. The microscope should be used to establish the diagnosis when there is doubt.

Induration, a hard, pearly border, and the absence of the miliary abscesses in the margin, are sufficient to differentiate an epithelioma from a blastomycotic lesion.

Technic of Examining for Blastomycosis in a Given Case.—The border is cleansed; then with a clean, sterile needle a droplet of sero-pus is removed from one of the miliary abscesses and placed on a clean slide. This is then covered with a drop of a ten per cent solution of potassium hydrate, a clean cover slip placed over the drop, which is examined after five minutes with a one sixth or one seventh objective. The double-contoured and usually budding organism of blastomycosis may be plainly recognized when present.

Prognosis.—Practically all cases of the purely cutaneous type recover in time under proper treatment. Recurrences, however, are common. Twelve of the fifteen patients with undoubted general blastomycosis have died. Only one has apparently entirely recovered. In all generalized cases, therefore, the prognosis is grave.

Treatment.—The chief remedial agent employed successfully is potassium iodid, first advised by Dr. Bevan. This drug nearly always produces marked results, and in many cases has entirely cured the disease. Doses as large as 600 grains per diem are often required. It should be given in large dilution. It may be gradually increased, as in syphilis, or large doses in large dilution may be given early. It should be administered until the last vestige of the disease disappears or while renewed activity occurs after its withdrawal. Radiotherapy is of value in completely eradicating small resistant areas. More recently Dr. Bevan has advised a trial of copper sulphate in one-quarter grain doses three times daily, with a one per cent solution applied as a wet dressing locally, with good results. In the grave, systemic cases, potassium iodid has exerted only inhibitory effects, except in one case, where it apparently had much to do with the recovery of the patient. It is interesting to note that this patient recovered under large doses of potassium iodid and a change of residence to California.

In cutaneous cases a surgical procedure is not indicated unless the entire lesion can be excised. Recovery has followed complete excision in a number of local cases. The large abscesses and other lesions occurring in the generalized cases require surgical interference.

LITERATURE.—*Gilchrist, T. C.* Johns Hopkins Hosp. Rep., 1896, Vol. I.—*Gilchrist-Stokes.* Bul. of Johns Hopkins Hosp., 1896, Vol. VII.—*Busse, Otto.* Cent. f. Bakt. und Parasitenk., 1894, XVI, p. 175.—*Busse-Buschke.* Virchow's Archiv, 1895, Vol. CXL, p. 23; Verhandl. der Deutschen Dermatologischen Gesellschaft, Schester Congress, 1899, p. 181.—*Curtis.* Ann. de l'Instit. Pasteur, 1896, Vol. X, p. 449.—*Wells, H. G.* N. Y. Med. Jour., March 26, 1898.—*Hessler, Robert.* Ind. Med. Jour., 1898, Vol. XVII, p. 48.—*Hyde, Hektoen,* and *Bevan.* Brit. Jour. Dermatology, 1898, Vol. XI.—*Hektoen.* Journ. Experimental Medicine, 1899, Vol. IV, Nos. 3 and 4.—*Owens, Eisendrath,* and *Ready.* Annals of Surgery, 1899, Vol. XXX.—*Murphy-Hektoen.* Journ. A. M. A., 1899, Vol. XXXIII, p. 1383.—*Anthony-Herzog.* Journ. Cut. and Gen.-Urin. Dis., Jan., 1900, 1.—*Coates, W. E.* Medicine, Feb., 1900.—*Baldwin, L. B.* Jour. A. M. A., 1900, Vol. XXXIV, p. 292.—*Brayton, A. W.* Ind. Med. Jour., April, 1900, and July, 1901; Jour. A. M. A., Feb. 1st, 1902.—*Montgomery, "Ricketts."* Jour. C. and G. U. Dis., Jan., 1901, Vol. XIX, p. 26.—*Dyer, Isadore,* Jour. C. and G. U. Dis., Jan., 1901.—*Stelwagon, H. W.* Am. Jour. of Med. Sci., 1901. Vol. CXXI, p. 176.—*Harris, F. G.* Am. Jour. of Med. Sci., 1901, Vol. CXXI, p. 501.—*Ormsby* and *Miller.* Jour. Cutan. Dis., March, 1903.—*Cleary, J. H.* Trans. Chicago Path. Soc., Vol. XI, No. 5, May 9, 1904, and Medicine, Nov., 1904.—*Eisendrath* and *Ormsby.* Jour. A. M. A., Oct., 1905.—*LeCount* and *Meyer.* Jour. Infect. Dis., 1907. Vol. IV, No. 2.—*Bassoe, Peter.* Jour. Infect. Dis., 1906, LLL, p. 91; Trans. Chic. Path. Soc., Vol. VI, No. 10, p. 380.—*Irons, E. E.,* and *Graham, E. A.* Jour. Infect. Dis., 1906, Vol. III, No. 4.—*Christensen* and *Hektoen.* Jour. A. M. A., July 28, 1906, Vol. XLVII, No. 4.—*Baum* and *Stober.* Demonstration of sections, Chicago Derm. Soc., April, 1907.—*Garvy, A. C.* Demonstration before a branch of Chicago Med. Soc., May, 1907.—*Montgomery, Frank Hugh.* Case demonstration before Chicago Derm. Soc., April, 1905. Jour. Cut. Dis., 1907, Vol. XXV.—*Hyde* and *Montgomery.* Jour. A. M.A., June 7, 1902, p. 1486, Vol. XXXVIII, No. 23.—*Ricketts, Howard T.* Jour. Med. Res., Vol. VI, No. 3.—*Evans, F. J.* Jour. A. M. A., June 27, 1903, Vol. XL, No. 26.—*Hamburger, W. W.* Jour. Infect. Dis., Vol. IV, No. 2, 1907.—*Otis, F. J.,* and *Evans.* Jour. A. M. A., Oct. 31, 1903, Vol. XLI, No. 18.—*Eastman, J. R.,* and *Keene, T. V.* Annals of Surgery, Vol. XL, No. 5, Nov., 1904.—*Wernicke* (quoted by *Gilchrist, T. C.*).—*Rixford, Emmeti, M.D.,* and *T. C. Gilchrist, M.R.C.S.* (Eng.), *L.S.A.* (Lond.), Johns Hopkins Hosp. Reports, 1896, I, 209.—*Ophüls, W.* Jour. Exper. Med., Vol. VI, Nos. 4, 5, 6.—*Ophüls, W.,* and *Moffett, H. C.* Philadelphia Med. Jour., June, 1900. *Ophüls, W.* Jour. A. M. A., Oct. 28, 1905, Vol. XLV, No. 18.—*Montgomery, D. W., Rykfogel, H. A. L.,* and *Morrow, H.,* J.C.D., Vol. XXI, p. 5, 1903.—*Bevan, A. D.* Jour. A. M. A., Nov. 11, 1905.

CHAPTER X

TUBERCULOSIS

THE organism now known as the tubercle bacillus was proven by R. Koch in 1882 to be the cause of tuberculosis. The discovery of this organism made clear a number of pathological changes affecting different tissues and viscera, and made possible the grouping of a number of diseases which formerly had been considered to be separate and distinct. Undoubted clinical examples have convinced us that

bovine tuberculosis may be transferred to man, in spite of the fact that Koch (1901) was unable to infect cattle and pigs with bacilli of human origin and expressed his belief that the converse—i. e., that the bacillus causing bovine tuberculosis was not pathogenic for man—was true. More recent investigations, such as those of Kossel, Weber, and Heusz, have shown that there are slight morphologic and cultural differences between the human and bovine types, that there are also some differences in pathogenicity, but that they are very closely related. Bovine bacilli are more virulent for most mammals than are human bacilli. Cattle are not susceptible to the latter, while pigs and goats develop, after subcutaneous injections, a chronic, progressive form of tuberculosis. Rabbits are less susceptible to the human than to the bovine type. There is no marked difference in pathogenicity for guinea pigs. Von Dungern has shown that the same results follow inoculation and feeding experiments performed upon anthropoid apes with these two types of bacilli.

The practical question is whether, after determining the differences between these two types of bacilli, the precautions taken against the transmission of bovine tuberculosis to man are superfluous. Later investigations have shown that they are not. Of course, the human type is found most frequently in tuberculosis as it occurs in man, but the bovine type is found also, especially in children. The latter type is found not only in the primary tuberculosis of the intestines and mesenteric lymph nodes, but also in the peritonitis which follows, in the viscera, in the miliary forms, and even in some cases in tuberculosis of cervical lymph nodes, of the joints, and of the skin following injuries. It may be easily seen that the bovine bacilli are highly significant as an etiological factor in human tuberculosis.

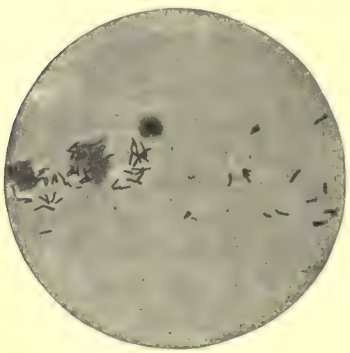

Fig. 154.—Tubercle Bacilli in Fresh Sputum.

Bacillus of Tuberculosis.—Tubercle bacilli are non-motile, often slightly curved, slender rods from 1.5 to 4 μ in length. They occur singly or in groups and clusters in the tissues, and, according to the present view, do not form spores. In animals under certain conditions, they grow out into longer or shorter branching threads, resembling actinomyces somewhat. Friedrich and Nösske observed this method of growth after intra-arterial injections of virulent cultures.

Resistance of the Bacilli.—The great resistance of tubercle bacilli explains the different modes of infection, in spite of the fact that they

do not grow outside of the body. They are not injured by drying or cold, and withstand 212° F. of dry heat for some hours. In the sputum they are not killed by chemical agents, as they are surrounded by mucus. Moist heat at a temperature of 203° F. kills them in from one to two minutes. In sputum they are killed by boiling for five minutes.

Cultures.—It is difficult to obtain tubercle bacilli in pure culture, for if the material is contaminated the other micro-organisms develop much more rapidly and overgrow them. Pure cultures are most easily obtained by transferring fresh tissues or a slightly caseated focus from an infected guinea pig to blood serum or glycerin-agar. There then develop after two or three weeks if the tubes are kept at a temperature of 98.5° to 100.5° F. with free access of air, small, white, dry scales, which later become confluent to form a membrane. Other growths upon ordinary agar and bouillon may be obtained by transplanting this pure culture.

Experimental Animals.—The guinea pig is best suited for experimental purposes, as it develops the disease most rapidly and in its severest form. An animal dies in from ten to twenty days of a general tuberculosis if a small amount of tissue containing bacilli or a pure culture is injected into the peritoneal cavity; after some weeks, following subcutaneous injections, localized nodules and ulcers having developed in the meantime. Rabbits are less susceptible and die of a general tuberculosis only after intra-venous or intra-peritoneal injections. Tuberculosis of the intestines, mesenteric lymph nodes, tonsils, and cervical lymph nodes have followed the feeding of pure cultures; tuberculosis of the lungs, the inhalation of powdered cultures; and tuberculosis of bones and joints (W. Müller, Friedrich), the injection of bacilli into the arterial system (femoral artery, aorta).

Methods of Staining.—There are a number of different methods for staining tubercle bacilli. It is one of a group of " acid-fast " bacilli, and does not readily give up the stain which it takes. Counter stains may be used and the bacillus may be readily differentiated from the surrounding tissues and associated micro-organisms. They stain not only by Gram's method, but also with aniline dyes, if an alkali, aniline oil, or carbolic acid is added and the stains are allowed to act for some time. Other micro-organisms and cells, present in smears or tissues, may be destained with alcohol or acids.

Examination of cultures, exudates, tuberculous débris, and sputum are made in the following way: Thin, even smears of the material to be examined are made upon cover glasses. The cover glasses are then passed through a flame two or three times until the smear is dry. Ziehl's carbol-fuchsin is then dropped upon the cover glass until the

smear is thoroughly covered. The cover glass is then gently warmed until steam arises; the specimen is allowed to remain in the hot stain two or three minutes, and is then washed in water. The other bacteria and cells are then destained by placing the cover glass for a short time in twenty-five per cent nitric acid, or, better, a three per cent solution in hydrochloric acid in alcohol. After washing in water the smear is counterstained with a dilute aqueous or alkaline solution of methylene blue. This solution is then removed with blotting paper and the preparation is again washed with water. Balsam may be applied to a well-dried cover glass and a permanent preparation made.

Staining of Bacilli in Tissues.—Ehrlich's mixture of a saturated aqueous aniline solution with an alcoholic solution of fuchsin or gentian, in which the sections should remain from twelve to twenty-four hours, and Ziehl's solution are to be recommended for staining the bacilli in tissues. After washing they should be destained in a three per cent solution of hydrochloric acid in alcohol, or in a twenty-five per cent solution of nitric acid and placed in sixty per cent alcohol. They should then be washed in water a number of times to remove the acid, and counterstained with methylene blue or Bismarck brown.

Differences between the Bacilli of Tuberculosis and of Leprosy and Smegma Bacilli.—For the method of differentiating the bacilli of tuberculosis and leprosy, *vide* page 448. The tubercle bacillus may be mistaken for the smegma bacillus (e. g., in examinations of the urine). Cornet recommends Weichselbaum's method for differentiating between tubercle and smegma bacilli. Stain with carbol-fuchsin, then, without destaining, use a concentrated solution of methylene blue in absolute alcohol. The tubercle bacilli remain red, the smegma bacilli become blue. Animal inoculation is, of course, the surest and most satisfactory method of differentiating between the two.

The difference in virulence of tubercle bacilli from different sources and during cultivation (decrease of virulence when growing upon media, and increase when passed through animals) depends upon the difference in the toxin-content of the bacilli (von Behring).

Toxins.—According to von Behring, toxins are found in the culture media and in the protoplasm of the bacilli. The latter (endotoxins) produce, when dead bacilli are injected into animals, inflammation, suppuration, and, when a thick emulsion is injected intravenously, tuberculouslike changes in the tissues (Koch, Masur, Kockel, and others).

Different toxic substances are found in the residue, which contains bacilli, obtained by the filtration of cultures. Koch prepared his tuberculin by making a glycerin extract of this residue. Von Behring analyzed this residue into separate substances, the end product of which, *tuberculosin*, he regards as the active and specific toxic base.

Immunization of Animals—Tuberculin-R.—Koch with his tuberculin was the first to attempt to immunize animals against tuberculosis and to cure animals already diseased. Tuberculous guinea pigs treated with tuberculin remained alive longer than control animals which were not treated (Pfuhl, Kitasato), but the animals never recovered, as Koch in the beginning thought possible. Tuberculin has not proven to be of any great value as a therapeutic measure. The changes in the local condition and the general reaction which follow its injection in tuberculous subjects make it very valuable for diagnostic purposes, although dangerous exacerbations and collapse may follow its use. It is used for diagnostic purposes, especially in cattle. When finally no reaction follows the use of tuberculin, because of immunization against it, the tuberculous process may extend or develop anew. The immunity which is established against tuberculin has no effect whatever upon the development and growth of the bacilli. In order to produce an immunity against the bacilli, Koch prepared his Tuberculin-R. [Tuberculin-R. is made in the following way: "Dried masses of the organism are ground up in an agate mortar. After suspension in distilled water and centrifugation, the emulsion consists of two layers. The overlying, opalescent, whitish fluid is designated as 'T. O.' (Tuberculin-Obers). After the removal of the fluid from the precipitate the latter was again dried and ground, suspended in water and centrifugated as before, and the process repeated until none of the sediment remained. The different fractions of fluid, except the 'T. O.' were combined to constitute 'T. R.' (Tuberculin-Rest), which is really an emulsion of minute fragments of bacilli."—Ricketts, "Infection, Immunity, and Serum Therapy," p. 413.] The pyogenic substances are removed by precipitation and then the tuberculin is rapidly absorbed and no abscesses form.

A. Wasserman and Bruck attempt to explain the occurrence or absence of the tuberculin reaction in the following way: According to their investigations, antituberculin is formed in tuberculous foci (local immunity), and the tuberculin injected into the body is attracted to the tuberculous foci by these antibodies. The complement is bound when the tuberculin unites with its antibody and there is a local accumulation of ferments which liquefy the tissues and produce softening of the diseased foci. There is no local reaction, likewise no general reaction, when, as a result of tuberculin treatment, antibodies are found in the blood; then the latter become bound to the tuberculin in the circulating blood, and the tuberculin is prevented from reaching the tuberculous foci. Von Behring (1902) successfully immunized cattle with the human type of bacillus, not dangerous for cattle, against bovine tuberculosis. Just as Jenner succeeded in immunizing man against small-

pox by inoculating him with cowpox, so von Behring was able to inoculate cattle with the human bacillus and render them immune against infection with bovine tuberculosis (therefore, Jennerization). The results obtained by von Behring have been confirmed by Baumgarten.

Modes of Infection.—Infection may follow the inhalation of dust or particles laden with bacilli, the ingestion of infected food, or the inoculation of wounds or ulcers. The disease has been transferred from a diseased mother to the fœtus.

Whether the disease develops or not after the bacilli have been introduced depends upon the number and virulence of the bacilli and the resistance of the patient.

Pulmonary tuberculosis is the most common form of the disease in man. It may follow directly the inhalation of the bacilli or may be secondary to a tuberculosis of lymph-nodes. Normally the upper respiratory passages are protected from infection by their mucous secretion, and for this reason are much more rarely involved than the lower. The sputum of a tuberculous patient carries with it the greatest dangers of infection in this form of tuberculosis. Bacilli are also found in the secretion of tuberculous ulcers, and are discharged into the outer world in the fæces and urine of tuberculous patients, but infection from these sources is not as common as that from the sputum. The sputum, unless proper precautions are taken, later dries upon the floor or pocket handkerchiefs, is pulverized and mixed with the air. Attempts have been made to educate the public not to spit upon the floors of large assembly halls, but into spittoons containing water, in order to prevent the dangers of infection (Cornet). The air about tuberculous patients may be infective, as small drops of fluid which contain bacilli are discharged when the patient coughs. The possibility of infection by the inhalation of both dried and moist tuberculous sputum has been demonstrated experimentally (Cornet, Flügge).

Tuberculosis of the gastro-intestinal tract may follow the ingestion of infected food and drink and the swallowing of tuberculous sputum. The pernicious habit some mothers have of licking the artificial nipple used upon the nursing bottle, and of chewing the bread before giving it to the child is a source of danger in this form of tuberculosis. The milk of cows suffering from a general tuberculosis or a tuberculosis of the udder is rich in bacilli, and may cause an intestinal tuberculosis. There is also danger of infection from eating meat from tuberculous cattle which is imperfectly cooked, and butter made from milk containing the bacilli. Roger and Garnier have demonstrated that the milk of a nursing mother may contain bacilli even when there is no disease of the breast, and, besides, the excreta of a tuberculous mother are always the source of grave danger to the child.

Finally, tubercle bacilli may be deposited upon recent and old wounds, gangrenous areas, and ulcers. Bacilli may gain access to wounds of the hands during operations, post-mortem examinations, the slaughtering of diseased cattle, or the milking of cows with tuberculous udders, and they may be carried on the hands to other parts of the body. Small wounds of the skin (scratches) or ulcers (ulcus cruris) may become infected in this way. Infection may follow an injury produced by an instrument or foreign body to which bacilli were attached. Occasionally infection may follow a fall, but this is rare, as the dust of the street, unless it contains fresh tuberculous sputum, is free from tubercle bacilli (Cornet). Secondary infection of a wound, because improperly treated (use of court plaster moistened with sputum, washing out and bandaging with infected handkerchiefs), is much more frequent. Tuberculosis of circumcision wounds, following attempts by a tuberculous rabbi to control hæmorrhage by sucking the bleeding surfaces, belongs to this class of infections.

Transmission of tuberculosis from the mother to the fœtus is possible (placental infection). Friedmann's experiments show that tubercle bacilli may pass with the spermatozoa into the ovum (conceptional and germinal transmission). Tuberculosis should be supposed to be of congenital origin only when it develops in the newborn, as older children living with tuberculous parents have been exposed since birth to infection. Experience shows that the children of tuberculous patients are more susceptible to tuberculosis than the children of non-tuberculous patients (hereditary predisposition), and also that those whose nutrition is not good or who have been weakened by previous disease are especially susceptible (acquired predisposition).

Histology of the Tubercle.—When tubercle bacilli are deposited in tissues they multiply slowly and incite a number of tissue changes. These begin with a degeneration of surrounding connective tissues and cells. This degeneration is then followed by a proliferation of the healthy connective tissue cells and the immigration of leucocytes into the area involved. Small grayish, cellular nodules, to which the name of tubercle has been given, are formed. Virchow (1852) suggested that the term *tubercle*, which before had had a general application, be applied only to this pathological change. The tubercles, which rarely become larger than a millet seed, compose the tuberculous granulation tissue which is produced by the proliferation of tissues in which the bacilli are deposited, and by the immigration of leucocytes. Occasionally tuberculous granulation tissue develops without tubercle formation.

The changes occurring in the tubercles and the tuberculous granulation tissue determine the course and the sequelæ of the disease.

According to Baumgarten's investigations, the connective tissues

and the endothelial cells of the blood and lymph vessels, sometimes the epithelial cells, react to the stimulus of the tubercle bacilli and their toxins by dividing mitotically. In about one week in animal experiments there is an accumulation of large cells (epithelioid cells) which resemble epithelial cells and develop mostly from fibroblasts. Leucocytes in different numbers wander from the neighboring vessels into the inflamed area (Fig. 155). If leucocytes are present in such large

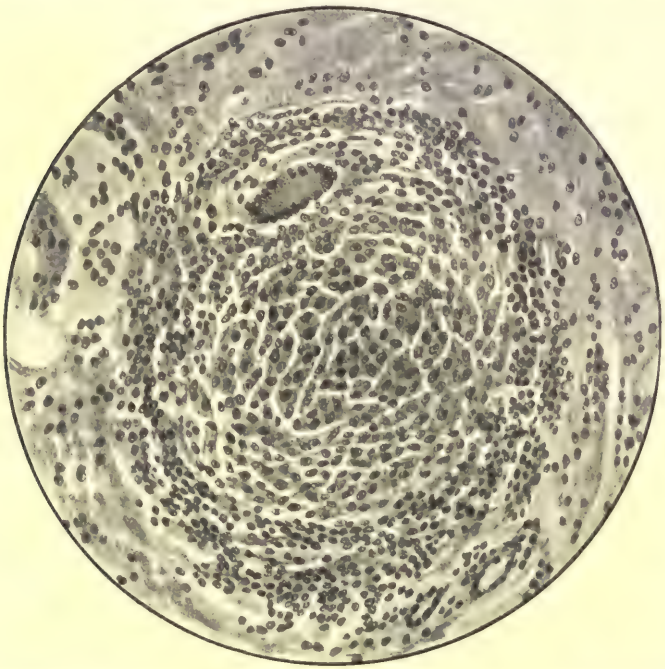

FIG. 155.—SECTION THROUGH A TUBERCLE. Upon the margin of the tubercle lymphoid cells may be seen; in the center epithelioid cells and a giant cell.

numbers that almost nothing can be seen of the large epithelioid cells, one speaks of a small-cell or lymphoid tubercle, in contradistinction to a large-cell or epithelioid tubercle, in which the large cells resembling epithelial cells predominate. The old connective tissue fibrils, separated by the cellular infiltration, form the supporting structure, the *reticulum* of the tubercle. In the center of the tubercle there are frequently found one or more multinuclear giant-cells (Langhans' type) in which the nuclei have a polar or peripheral arrangement. Groups of bacilli may be found within the cytoplasm of these cells. It is supposed that the bacilli stimulate nuclear division in these cells, and that the failure of cell division is due to the injury of the cytoplasm. [" Metschnikoff and others take a different view of the formation of giant-cells, con-

sidering that they represent individual epithelioid cells which have fused to form a multinuclear mass.''—Ricketts, '' Infection, Immunity, and Serum Therapy.'']

Regressive Changes in the Tubercle.—The fully developed tubercle, which is gray, translucent, and may be as large as a millet seed, begins sooner or later, under the toxic action of the bacilli, to degenerate in the center. Round cells, fibroblasts, and giant-cells gradually degenerate until the entire tubercle is transformed into a hyaline (coagulation necrosis of Weigert), finally into a granular, fatty mass, in which the bacilli gradually die. The caseated tubercle is opaque and yellowish-white. A fibrous capsule is formed by the proliferation of the surrounding connective tissue which may replace entirely or partly the degenerated mass (fibro-caseous tubercle).

The tubercle or groups of tubercles may undergo a number of regressive changes. The tubercle imbedded in the tuberculous granulation tissue has upon section the appearance of a spongy, semitransparent, grayish red (if caseation occurs, yellowish) mass. Ulcers and fistulæ follow regressive changes in tubercles situated near the surface of the body, in the skin, or mucous membrane. Large caseous (if the connective tissue proliferates, fibro-caseous) nodules follow the degeneration of tubercles situated in the deeper tissues or viscera. If the

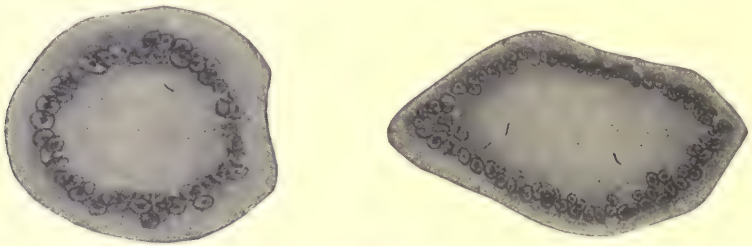

Fig. 156.—Tuberculous Giant Cells Containing a Few Tubercle Bacilli.

tubercles become softened and liquefied, large cavities with contents resembling pus (cavities in the lung, abscesses in lymph nodes) are formed. The extension of the tuberculous process and the development of tuberculous (cold) abscesses follow the deposition of bacilli in surrounding tissues and the development of new nodules. Gravity and the anatomical arrangement of the loose, fascial planes are important in determining the direction in which the disease extends and tuberculous pus burrows (gravitation abscess). In ulcers of the skin and mucous membranes the tubercles lie exposed in the pale, flabby, glassy, or yellowish granulations; they cover the synovial membrane in tuberculous arthritis, and are found in the walls of tuberculous abscesses (Fig. 157).

The pathological changes, formation of granulation tissue, caseation, suppuration, or encapsulation, vary in the different forms of tuberculosis. The virulence of the bacilli and the resistance of the tissues determine whether the tuberculous foci will cicatrize, caseate, or suppurate. The first form, which König has called the dry tuberculous granulation tissue, is much more benign than the caseating form. This difference in clinical course seems to depend upon whether small or large amounts of toxins are formed. In tuberculosis of the serous cavities and joints, the pathological processes are still more complicated, as a serofibrinous exudate is formed.

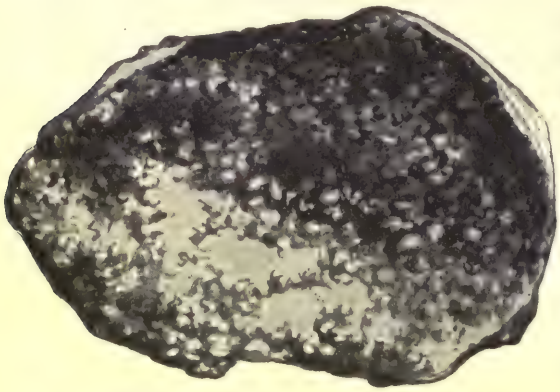

FIG. 157.—TUBERCULOUS ABSCESS MEMBRANE.

Secondary Infection.—If a tuberculous focus becomes infected with pyogenic bacteria, through the blood or a fistula, an acute inflammation develops and a purulent exudate is formed as the result of the secondary pyogenic infection.

Tuberculous Pus.—Tuberculous pus, the result of liquefaction of caseous material, differs from that formed in suppurative inflammations caused by pyogenic bacteria. As the caseous material liquefies, it becomes mixed with a serous or serofibrinous exudate. Tuberculous pus is watery, white or light green in color, and contains masses of caseous material, strings and flakes of fibrin. On the other hand, pus formed in suppurative inflammations is thick and creamy, as the solid particles are evenly distributed throughout its substance. Pus corpuscles in tuberculous lesions early undergo fatty degeneration and disintegrate (Tavel), so that but few are found when the pus is examined microscopically. Tubercle bacilli are not numerous, and it may be impossible to demonstrate them by microscopical examination. Often it is necessary to resort to animal inoculations to determine the character of pus removed from doubtful lesions. The liquefaction of tuberculous nodules and infiltrations is due to the action of polymorpho-nuclear leucocytes, and not to that of tubercle bacilli or pyogenic bacteria. When the leucocytes disintegrate ferments are set free, which digest and liquefy the necrotic, caseous material (Friedrich Müller).

A local, limited tuberculosis is differentiated from a general miliary tuberculosis, which develops from the former. The majority of the lesions developing in localized, limited tuberculosis are treated surgically.

LOCAL SURGICAL TUBERCULOSIS

The foci may develop in the infection atria or in the parts where the bacilli are deposited by the blood and lymph streams.

(a) TUBERCULOSIS OF THE SKIN

The Anatomical Tubercle.—The so-called "anatomical tubercle" may develop upon the hands of individuals who work with tuberculous materials from man or cattle, following an injury of the skin. A small, firm, reddish brown nodule, which may become as large as a pea, develops at the point of injury after some weeks. It is covered with a cornified, fissured epidermis, and often, after lasting for some time, disappears spontaneously. The "anatomical tubercle" is the most benign form of tuberculosis of the skin. It does not ulcerate, and only rarely is it followed by involvement of glands about the elbow. Mixed infections occur frequently.

Verrucous Tuberculosis of the Skin.—According to Riehl and Paltauf, verrucous tuberculosis of the skin (tuberculosis cutis verrucosa) is a form of tuberculosis which also follows injuries of the skin. The flat, slightly elevated, usually round, infiltrated area has a bluish red border and an irregularly warty surface. It pursues a chronic course, and the infiltrated area, without ulcerating, becomes as large as a silver dollar or may finally involve the dorsum of the hand and a part of the forearm. The hands are most frequently attacked, especially in such people as butchers and those who come in contact with materials from tuberculous cattle. A case in von Bergmann's clinic followed an injury of the dorsum of the hand with a milk pail. The epitrochlear and axillary lymph nodes may be involved after the skin lesion has persisted for some time.

Tuberculous ulcers of the skin, excepting those developing in lupus exulcerans, are most often secondary to a tuberculosis of the mucous membranes of the mouth, rectum, and genitalia. The process as a rule extends from the mucous membrane to the skin. These ulcers usually develop in the terminal stages of some of the other forms of the disease from small miliary nodules which form in the skin. The ulcers are characterized by flat, irregular, undermined borders, and by pale, translucent, yellowish, soft granulations which may be easily wiped away with little hæmorrhage with gauze.

It has already been mentioned that the wounds following circum-

cision in young Jewish children may be infected, as a result of the sucking of the wound by a tuberculous rabbi. Small nodules then develop in the prepuce, which later ulcerate and lead to the formation of chronic ulcers. After some weeks the inguinal lymph nodes become involved; general glandular and miliary tuberculosis have been observed.

Lupus.—Tuberculosis of the skin develops most frequently in the form of small brownish red macules or slightly elevated nodules, which are sometimes hard. The nodules may be of pinhead size or may become as large as a pea. The color of the nodule disappears but little when pressure is made; this is an important diagnostic sign. They develop in all layers of the cutis, frequently also in the subcutaneous fat, and correspond to the miliary tubercles or to a number of the same. They may be single or multiple, and the area of skin involved may be very circumscribed or extensive.

FIG. 158.—LUPUS EXULCERANS AND EXFOLIATIVUS FACIEI.

The face is most frequently affected in this form of cutaneous tuberculosis, the so-called lupus. In 100 cases the disease attacked the face 76 times, and of the 76 cases the nose was affected in 38. The skin of the extremities is attacked relatively frequently. The development of lupus nodules has been observed after inoculation of the skin with tubercle bacilli—e. g., after the use of saliva in tattooing. There are a number of cases in which the ectogenous infection of an already diseased area of skin (eczema) has been demonstrated. According to Cornet it is not essential that the skin be injured, for tubercle bacilli, like staphylococci, may be forced into the hair follicles and deeper tissues by rubbing (e. g., wiping the nose with an infected handkerchief). Of course the bacilli may be carried in the patient's sputum, and the skin about the mouth may become infected from it. The skin surrounding a tuberculous fistula or covering a caseated lymph node

or a tuberculous focus in a bone, may become secondarily involved. Tubercles may develop at the muco-cutaneous margins (lips, nose, and anus). The infections at these places may develop from the lymphatics,

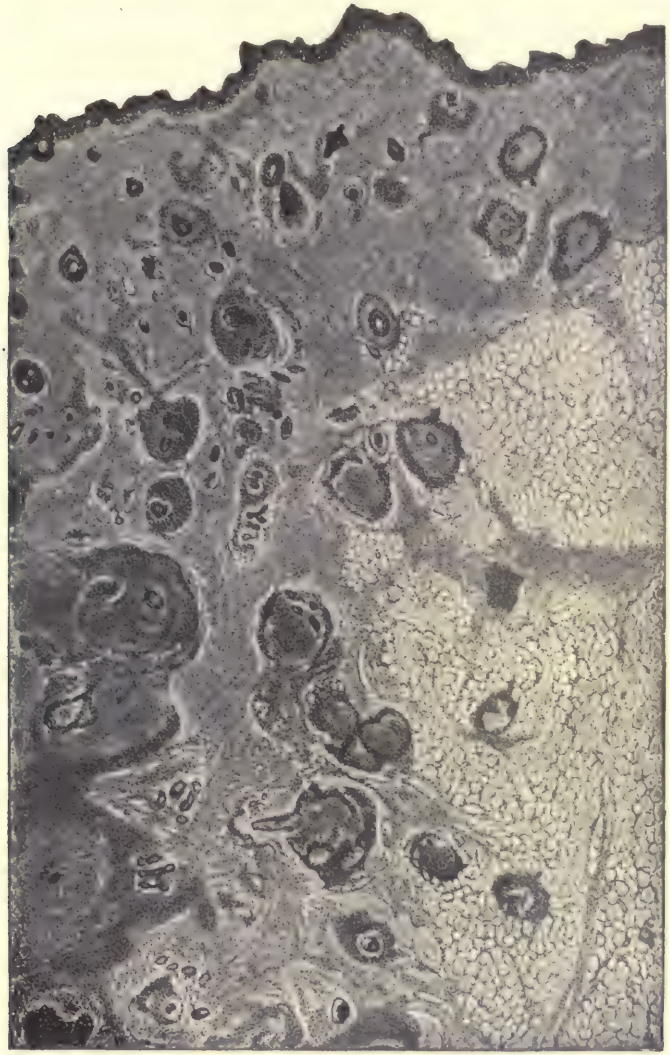

FIG. 159.—CUTANEOUS TUBERCULOSIS (LUPUS FACIEI). Tubercles may be seen in the subcutaneous fat.

or may be the result of direct infection with the secretions which contain bacilli. The classical form of lupus of the face is frequently secondary to a lupus of the nasal mucous membrane. It begins in the nose and then extends to the skin of the nose, upper lip, and both

cheeks, producing the well-known "butterfly-shaped" lesion. Sometimes tuberculosis develops in the scar resulting from extensive operations for tuberculosis of bone (e. g., joint resection), and sometimes in these cases bacilli are carried into the skin during the operation; at other times the wound is infected by the secretion discharged during the repair of the wound. As compared to the ectogenous and lymphogenous infections, hæmatogenous infections are rare.

Different Clinical Pictures.—Tuberculosis of the skin presents different clinical pictures, depending upon the area of skin involved, the size of the nodule, and whether the progressive or regressive predominate. Sometimes the nodules disappear spontaneously and healing occurs; sometimes the nodules ulcerate; at other times the tissues hypertrophy, producing large disfiguring growths. The following forms of lupus are described: Disseminated lupus (lupus disseminatus), in which single nodules are scattered throughout the area involved; serpiginous lupus (lupus serpiginosus), in which the nodules are close together and arranged in the form of a curve; exfoliating lupus (lupus exfoliativus), with exfoliation of the skin, extrusion or absorption of the caseated tubercle, and subsequent healing; ulcerating lupus (lupus exulcerans), in which the larger nodules ulcerate, forming shallow ulcers, the process often extending at the periphery, while the center heals, deformities of the face, toes, and fingers following the cicatricial changes; hypertrophic lupus (lupus hypertrophicus), in which there is a marked growth of the cutaneous and subcutaneous connective tissues (occurring particularly upon the lobule of the ear); papillary or verrucous lupus (lupus papillaris or verrucosus), resulting from a growth of the papillæ of the skin; and lupus cornutus, in which the epidermis becomes cornified.

FIG. 160.—HYPERTROPHIC AND ULCERATING LUPUS. Healing after excision of lesion and transplantation of a large cutis graft from the thigh.

Clinical Course of Lupus.—The clinical course of lupus is chronic. Frequently (in eighty-five per cent of the cases) tuberculous foci

develop in the viscera, bones, or joints. The process subsides for a time in the skin involved, and then develops again, this process occurring repeatedly in the course of the disease. The caseated nodules are absorbed or extruded and are replaced by scar tissue, but soon new tubercles develop in the scar or in the tissues surrounding it. The mildest cases are those in which the nodules do not coalesce and do not ulcerate. If, on the other hand, large areas of skin become infiltrated, deep ulcers may develop and large disfiguring scars may subsequently form. These scars produce the worst cosmetic and functional results when they involve the face, the fingers, and the toes. Scars upon the face may produce an ectropion of the lids or lips, a narrowing of the mouth, adhesions about the entrance to the nose. Lupus about the nose may destroy the alæ nasi with their cartilages. The concha may be destroyed in the same way. Scars upon the fingers and toes may produce contractures which destroy completely the function of the digits.

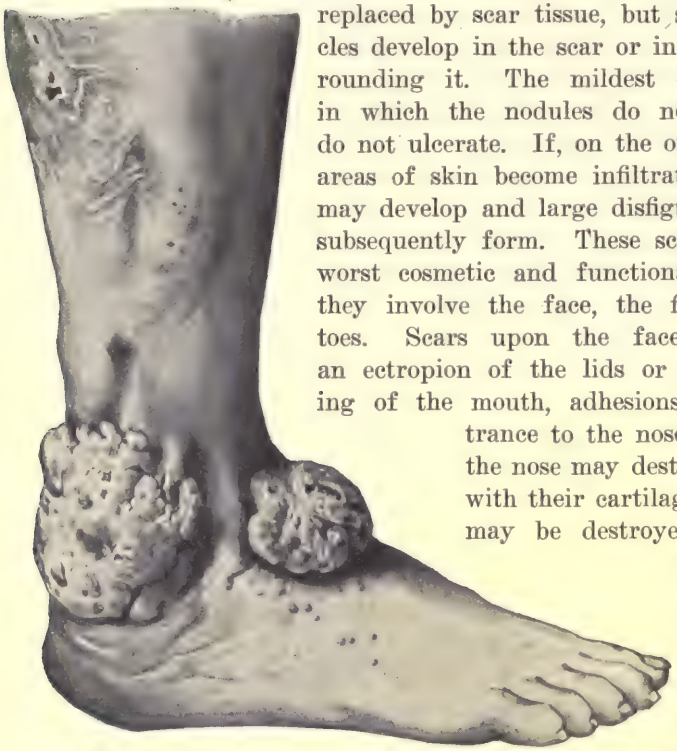

FIG. 161.—PAPILLARY TUBERCULOSIS OF THE SKIN.

The lymph nodes adjacent to the area involved are almost always involved in lupus. Erysipelas frequently develops in the lupus ulcers (habitual erysipelas of lupus patients). This acute inflammation of the skin has a healing effect upon the lupus, for the infiltration may degenerate and disappear by absorption after such an infection.

A carcinoma may develop upon a lupus. The beginning of a carcinoma is indicated by the darker color and induration of the area involved.

Treatment.—Complete excision of the area involved, together with the subjacent fat, is the safest treatment, provided the lupus is circumscribed. It should be remembered that frequently single tubercles are found in the most superficial layers of the subcutaneous tissues. The resulting defect should be covered by skin-grafting. It is best to use in grafting large cutis grafts, for the epidermal strips shrink

when used upon the face and hand, and, besides, keloids frequently develop after removal of these tuberculous lesions.

Excision is impossible when the lesion is extensive—for example, if it involves all the skin of the face. The diseased tissue should then be removed with the sharp spoon as recommended by Volkmann. After the hæmorrhage is controlled, separate remaining islands of granulation tissue should be destroyed with the Paquelin cautery. If Hollaender's hot-air apparatus is employed without previous curettage, a beautiful scar develops after the eschar is detached, but soon new nodules develop, as the hot air has but a superficial action.

Plastic operations (rhino-, cheilo-, and blepharo-plasty) should not be attempted until the lupus has completely healed.

Koch's tuberculin is no longer used in these cases, as it is dangerous. It produces a severe reaction in the diseased area, resembling erysipelas clinically. Transitory healing follows its use, as the tuberculous tissue is rapidly destroyed. After the patient becomes immunized against the tuberculin there is no longer any local reaction, and new tubercles develop in the scar.

The X-ray and Finsen's light therapy appear to give the best results and should be regarded as the treatment of choice in most cases.

(b) TUBERCULOSIS OF SUBCUTANEOUS TISSUES

(*Scrofulodermia—Tuberculous Gumma*)

This form of tuberculosis develops most frequently in children as circumscribed nodules in the subcutaneous tissues covering tuberculous foci in lymph nodes and bones. Occasionally it develops in adults along the course of the lymphatic vessels of the extremities which drain a lupus. The skin covering the nodule becomes bluish red in color, and after the contents of the nodule soften and the *débris* ruptures through the skin, large chronic ulcers, in the floor of which tuberculous granulation tissue may be seen, develop.

Excision, curettage with the sharp spoon, tamponing with iodoform gauze, and removal of the primary focus are indicated in the treatment.

(c) TUBERCULOSIS OF MUSCLE

Tuberculosis of muscle is, as a rule, secondary to a deep ulcer of the skin or mucous membrane (e. g., on the cheek, lips, or tongue), or follows the rupture of a tuberculous focus situated in a bone, joint, or lymph node. Usually the process is a chronic inflammation of the interstitial substance of the muscle, associated with the formation of tubercles and subsequent destruction of the contractile substance. Abscesses develop when the larger masses of granulation tissue become

caseated and liquefied. When these are incised or rupture spontaneously, large amounts of cicatricial tissue develop in the muscle.

As compared to these secondary forms, the so-called primary hæmatogenous tuberculous myositis (myositis tuberculosa) is rare. This form of tuberculosis may produce large, fungous, and caseous foci or abscesses in the muscle which should be incised and thoroughly curetted and tamponed with iodoform gauze (Lorenz).

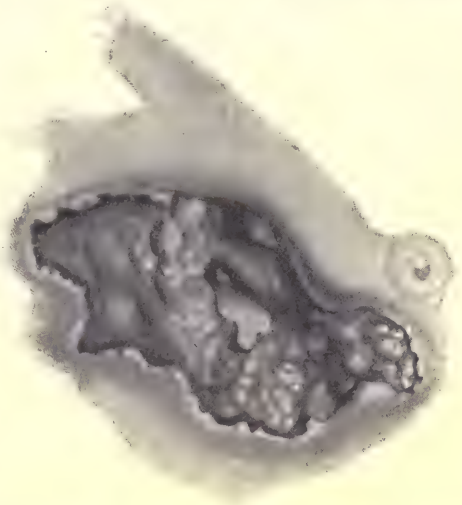

Fig. 162.—Tuberculous Ulcer over a Diseased Rib.

(d) TUBERCULOSIS OF THE MUCOUS MEMBRANES

Tuberculosis of the mucous membranes of the gastro-intestinal tract and upper respiratory passages is most frequently secondary to a tuberculosis of the lungs, the infection being carried in the swallowed or expectorated sputum. A hæmatogenous infection of mucous membranes is possible, but rarely occurs.

Tuberculosis of the Mucous Membrane of the Mouth, Lips, and Tongue.—The relatively rare primary infections of the mucous membrane of the mouth, lips, tongue, and nose may develop after slight injuries, if large enough numbers of bacilli are introduced in the food or air, as they easily become attached to areas deprived of their epithelium. Injuries of the mucous membrane of the mouth by pointed, carious teeth, of the mucous membrane of the nasal cavity by the finger nail, by which bacilli may be introduced at the same time, are important factors in this form of infection. If infection occurs, small miliary nodules or nodular infiltrations develop, from which extremely painful ulcers may result which may be superficial or deep. These ulcers have a granular, often yellowish, floor, and undermined, corroded edges which are not indurated as in carcinoma. It is not difficult to make a diagnosis in these cases, as small nodules and ulcers are present in the surrounding tissues.

Pharyngeal Tuberculosis.—A disseminated tuberculosis of the mucous membrane of the pharynx frequently occurs in advanced cases of tuberculosis of the lungs. In lupus of the face a disseminated tuberculosis of the lips and gums is rarely absent, occurring either as a primary or

secondary infection. The mucous membrane is reddened and granular and bleeds easily when touched, if regressive changes have occurred and the nodules have been transformed into irregular, painful ulcers.

Tuberculosis of the Tonsils.—The palatal and pharyngeal tonsils, because of their structure and the friction to which they are exposed in the first act of swallowing, are especially apt to become infected with bacilli which are contained in the sputum and particles expectorated by tuberculous patients. Primary infection by inhalation, by the ingestion of infected food, and by saliva with which a tuberculous mother moistens an artificial nipple has been observed. This form of infection is especially frequent in children, in whom the tonsils may be the source of an extensive glandular tuberculosis of the neck. Tuberculosis of the tonsils cannot be diagnosed unless ulcers are present. Usually it can be made microscopically only by the demonstration of tuberculous changes in the extirpated hypertrophied tonsils. Tuberculosis of the Eustachian tube and middle ear may follow a tuberculosis of the mucous membrane of the nasopharynx.

Laryngeal Tuberculosis.—The larynx is likewise frequently involved secondary to tuberculosis of the lungs; the rare primary infection may follow the inhalation of tubercle bacilli when the mucous membrane is irritated or chronically inflamed. The nodules and ulcers which develop upon the vocal cords are frequently mistaken for carcinoma.

Intestinal Tuberculosis.—The mucous membrane is most frequently involved in intestinal tuberculosis, especially at those points where there are lymphatic follicles. The sputum swallowed by tuberculous patients is the most important etiological factor. It contains large masses of bacilli which are not destroyed in the stomach, and the mucous membrane of the intestines is thus constantly exposed to infection. About ninety per cent of all cases of tuberculosis of the respiratory passages are accompanied by the intestinal form of the disease. Primary intestinal tuberculosis has been demonstrated in but a few cases (Cornet). Tuberculous intestinal ulcers are usually multiple. They extend along the course of the lymphatics and are circular as a rule. These ulcers may cause a perforation of the intestinal wall, or if they heal, a cicatricial stenosis. Ileo-cæcal tuberculosis is frequently of the hyperplastic type, and large masses are formed which may be mistaken for carcinoma. Ulcers and fistulæ not infrequently develop when the rectum is involved.

Treatment.—Tuberculosis of mucous membranes is rarely limited to a small area, and radical treatment, excision of the accessible lesions, can be employed in exceptional cases only. Cauterization with fifty to eighty per cent lactic acid or an eight per cent solution of zinc chlorid are the best known methods. Nodules and deep ulcers may be curetted

with a sharp spoon or cauterized with the actual cautery. Fistulæ in ano should be treated by the methods advised in operative surgeries. Resection or anastomosis may be indicated in the treatment of stenosis following tuberculosis of the intestine. The intestine should be resected, if possible, in hyperplastic tuberculosis. If resection is contraindicated, a lateral anastomosis between the ileum and ascending colon may be made to exclude the diseased bowel.

(e) TUBERCULOSIS OF LYMPHATIC VESSELS AND NODES

Lymphatic nodes adjacent to tuberculous foci are almost always diseased. Orth, Wesener, and Cornet have shown that tubercle bacilli may pass through mucous membranes without producing any pathological changes, and the last has also demonstrated that they may even pass through the uninjured skin. Not infrequently lymphatic nodes become infected when there are no pathological changes found in the mucous membranes which they drain (e. g., tuberculosis of mesenteric and cervical lymph nodes in children). The findings of the authors above mentioned explain how this can occur. The nearest node retains the bacilli, which multiply and then pass to adjacent nodes. The gradual extension of the infection from node to node may be followed especially well in animals (Cornet).

Tuberculosis of Lymphatic Vessels.—Tuberculosis of the lymphatic vessels is rare, if those cases are excluded in which the thoracic duct (in miliary tuberculosis) or the chylous vessels, which may be traced through the mesentery to the receptaculum chyli, are involved secondary to tuberculosis of the intestines. In rare cases a tuberculous lymphangitis develops in the extremities, most frequently in the upper (Jordan). The symptoms are pronounced only when the superficial lymphatics are involved, especially when there are tuberculous ulcers upon the hand or foot, or ulcers and fistulæ, which have followed the rupture of an osteal focus. In this form of tuberculosis one or a number of small nodules from which ulcers or larger subcutaneous nodules may develop form along the lymphatic vessels. The nodules may soften and discharge externally. Large, cordlike infiltrations, which later caseate, have been observed along the course of the lymphatic vessels, and when softening occurs, abscesses, fistulæ, and deep ulcers develop. A provisional diagnosis of tuberculosis of the deep lymphatic vessels may be made, if an abscess which has no connection with bone or a joint develops in the course of the larger lymphatic vessels.

Tuberculosis of Lymph Nodes.—Tuberculosis of lymph nodes (lymphadenitis tuberculosa) may develop at any age, but is most frequent between the fifteenth and twenty-fifth years (Fischer).

The lymphatic nodes become enlarged when infection occurs. This

enlargement is due partly to the increase in the number of cellular elements; chiefly, however, to the development of tubercles. These develop throughout the whole node and fuse so that sometimes large caseous foci rapidly develop (*vide* Fig. 164) and soften, so that abscesses, which may include all the glandular tissue, form. A caseous, purulent periadenitis develops as the process extends, and the nodes then become firmly adherent to surrounding structures (other nodes, muscles, large vessels, etc.). The caseous masses may rupture through the skin if a tuberculous abscess does not develop. An acute general miliary tuberculosis follows the rupture of a caseated cervical lymph node into the internal jugular vein, or the rupture of a caseated bronchial lymph node into the pulmonary vein. If the caseous material becomes inspissated and calcified, small round calcium concretions may be found (especially in the bronchial and mesenteric lymph nodes).

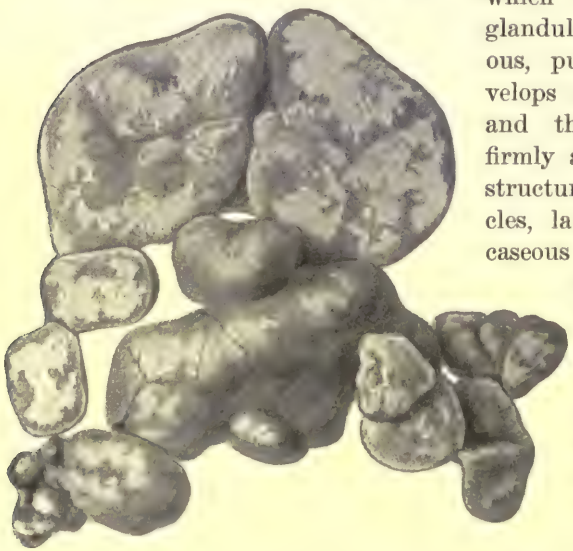

FIG. 163.—GROUP OF TUBERCULOUS LYMPH NODES REMOVED BY OPERATION. Caseous foci may be seen upon the cut surface of the nodes and also showing through the capsule.

The less the caseation, the greater the hyperplasia with the development of large epithelioid cells is and the longer the disease remains confined to single nodes, which may attain a large size. In the hyperplastic form of tuberculosis the nodes become firm and solid, and but little or not at all adherent to surrounding structures (non-caseating, indurated form, large-cell hyperplasia of lymph nodes of Ziegler).

In rare cases the nodes may undergo a pure hyperplasia, in which no or but few tubercles develop. In this form of tuberculosis a large number of widely separated chains of lymph nodes may become diseased. Clinically, it resembles closely pseudoleukæmia (aleukæmic adenie), and can only be differentiated from the latter by the demonstration of tubercle bacilli in the lymph nodes.

Tuberculosis of the cervical lymph nodes is most important surgically. They are most frequently involved, about ninety per cent (Fischer) of the cases of tuberculosis of lymph nodes occurring in them. The disease most frequently begins in the nodes of the submaxillary

triangle, as all the lymphatic vessels which drain the face, mouth, and pharynx unite to empty into these. In descending infections (originating usually in the mouth and pharynx) the nodes which lie along the great vessels are involved after the submaxillary and submental nodes. These nodes extend along the internal jugular vein down to the level at which it joins with the subclavian. The disease extends from the nodes beneath the sterno-cleido-mastoid muscle to those lying in the posterior triangle of the neck, a little anterior to the border of the trapezius. In ascending infections, which develop from a focus in the apex of the lung, the lymph nodes in the supraclavicular fossa become enlarged first.

When the primary focus is about the external canthus of the eye, the frontal or temporal region, the nodes superficial to or within the parotid gland become diseased; when in the cheek, the nodes lying upon the buccinator muscle.

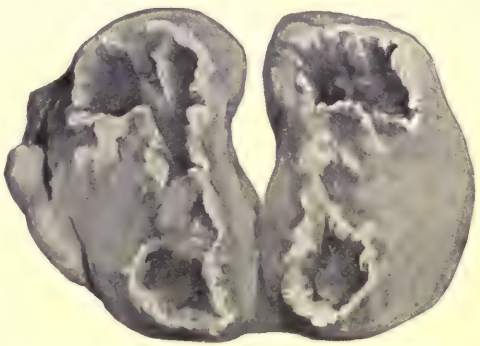

Fig. 164.—Incised Lymph Nodes, Showing Softened Caseous Foci.

A group of nodes is found in the axillary fossa beneath the pectoral muscles, which extends along the subclavian vessels to communicate with the supraclavicular nodes. The disease may extend along this chain from a focus below, or the reverse may happen, a tuberculosis may extend from the neck along this chain into the axillary fossa. When the primary focus is in the skin or bones of the hand, the epitrochlear nodes become diseased first.

The inguinal lymph nodes may become diseased secondary to a focus in the skin of the legs, about the anus, or upon the penis. When the disease involves the skin of the leg, the nodes in the popliteal fossa may become diseased first.

Tuberculosis of the mesenteric and retroperitoneal lymph nodes follows a tuberculosis of the intestines. In children these nodes frequently become tuberculous, even when there is no demonstrable lesion in the mucous membrane.

Tuberculosis of the bronchial lymph nodes is frequently important from an etiological viewpoint. It may be the origin of hæmatogenous infections of bone, joints, kidneys, and of miliary tuberculosis.

Diagnosis.—It is difficult to make a diagnosis early, when only single nodes are involved and there is no demonstrable primary focus. Tuber-

culous nodes are harder than those undergoing simple inflammatory hyperplasia. Sometimes they are so hard and firm that they resemble carcinomatous lymph nodes, and this should be kept in mind in making a differential diagnosis.

The gradual but progressive increase in the size of the nodes, and the involvement of an entire chain are important in making a diagnosis. The diagnosis is not difficult when the nodes soften, when periadenitis, abscesses, and fistulæ develop. Then the outlines of the separate nodes are no longer distinct, and they become attached to the surrounding structures and to each other. The development of tuberculous granulation tissue about the openings of fistulæ, and the finding of caseous *débris* in the pus make the diagnosis certain.

The rapid enlargement of the nodes when abscesses are forming, and the adhesions which they form with neighboring structures may remind one of lymphosarcoma. Recently diseased, small, hard, movable glands adjacent to the softened mass reveal the true nature of the process.

It is not always possible to differentiate between tuberculosis and malignant growths of the lymph nodes of the mesentery, unless the general course of the disease reveals the nature of the pathological process.

An examination of the blood excludes leukæmia. Pseudoleukæmic glands are movable upon one another, and as the disease does not extend beyond the capsule they do not contract adhesions with neighboring structures. A small softened node in the cheek, adherent to the skin, may be mistaken for an atheroma; in the neck, if the skin covering is not adherent, for a dermoid or a branchial cyst. Syphilitic enlargement of the lymph nodes is indicated by the ordinary symptoms of this disease.

Treatment.—An attempt should be made in the treatment to remove all the diseased lymph nodes and the primary focus when possible. The general condition of the patient should also be improved. Movable glands may be enucleated by a finger or blunt dissection from the surrounding structures. Adherent groups of nodes should be dissected free with knife and scissors. It is impossible to perform these operations unless one has an accurate knowledge of the anatomy of the part, for important nerves (in the neck, for example, the spinal accessory may be imbedded in the nodes) or large veins (internal jugular) may be so adherent that resection may be necessary. The large wounds resulting from extensive radical operations should be drained, the drainage being inserted at the lowest angle of the wound, and even graduated pressure should be exerted by the bandage so that all dead spaces will be rapidly obliterated.

The disease may recur in glands left behind. This, however, should not deter the surgeon from doing a radical operation for a majority of these cases recover when proper treatment is instituted. The statistics concerning the results of the radical operation in the treatment of tuberculous glands of the neck are as follows: fifty-four per cent of permanent recoveries, twenty-eight per cent of recurrences, eighteen per cent terminate fatally. [The immediate mortality from even extensive operations for the radical removal of tuberculous glands is surprisingly low. In the statistics given above eighteen per cent of the cases of the disease were not arrested, and death was the result of the involvement of other organs, and not the direct result of the operation.]

Contra-indications to the Radical Operation.—The radical operation is contra-indicated when the general condition of the patient is poor, when the glands are very adherent, and when a number of fistulæ have formed. In these the operation to be performed depends upon conditions; fistulous tracts should be curetted, abscesses incised, and the caseous masses and the abscess membrane removed with a sharp spoon. Large groups of nodes may be made to soften by injecting chemicals (Lugol's solution, carbolic acid and sublimate solution, iodoform-glycerin emulsion, etc.) into them. The abscess following softening should be incised and curetted with a sharp spoon or aspirated. The contents of large cold abscesses should be aspirated and iodoform-glycerin emulsion should then be injected (cf. Psoas abscess).

Fig. 165. — Illustrating the Condition which was Formerly Known as Scrofula. Enlargement of the nodes of the neck and cheek (lymphadenitis tuberculosa), inflammation and rhagades of the upper lip, chronic nasal catarrh, conjunctivitis.

Scrofula.—By scrofula is understood a disease of children in which there is a chronic catarrh, eczema, and inflammatory swelling of the mucous membrane of the lips, eyelids, and cheeks, associated with the enlargement of the lymph nodes, especially of those of the neck. This enlargement of the lymph nodes is caused by tubercle bacilli, which gain access to the lymphatics through the diseased skin and mucous membrane.

Some of these cases are not tuberculous, the lymph nodes enlarging as the result of the absorption of pyogenic bacteria or their toxins from the inflamed skin or mucous membrane. Sometimes the nodes become hyperplastic, sometimes they suppurate. This second form, which Cornet has called the pyogenic to differentiate it from the tuberculous form of scrofula, may become tuberculous, as the conditions which favor infection with tubercle bacilli are present and the resistance of the nodes is reduced.

Cases in which there is a tuberculosis of lymph nodes, bones, and joints should no longer, as was formerly done, be classified as scrofula.

(f) TUBERCULOSIS OF BONE (TUBERCULOUS OSTEITIS)

Tuberculosis of bone occurs chiefly as a tuberculous osteomyelitis, the bacilli being carried by the blood from some other focus, such as a tuberculosis of the lungs, lymph nodes, etc., although there are cases in which no primary focus can be demonstrated even by a post-mortem examination (König). The disease may extend from a joint and involve the bone secondarily. The possibility of infection through the lymphatic channels which drain an adjacent diseased area cannot be excluded.

The bone involvement occurring in acute general miliary tuberculosis is of no surgical significance.

Embolic Origin of Tuberculosis of Bone and Distribution of Arteries.—Apparently most of the tuberculous foci in bone are caused by the lodgment of infected emboli (particles of caseous matter or groups of bacilli, the so-called bacterial emboli). The shape of the foci, which is that of an infarct, certainly indicates that this is so. W. Müller has reproduced these changes experimentally by injecting tuberculous pus into the arteries of the extremities. Emboli passing from the right heart into the lungs produce severe symptoms of lung infarcts. Particles of emboli may pass into the radicals of the pulmonary vein and be carried to the left heart and from there into the general circulation. It is also conceivable that a focus in the lungs or bronchial nodes may rupture into a pulmonary vein, and that a general infection (*vide* Miliary Tuberculosis) may develop in this way.

The round, irregular foci and sequestra must frequently be of embolic origin. A study of the distribution of the blood vessels in developing long bones shows that an embolus passing in either the epiphyseal, metaphyseal, or nutrient arteries will lodge near the epiphysis, which is the favorite site for the development of the disease (*vide* Figs. 166 and 167). Only the large emboli lodge in the diaphysis of long bones, for the diameter of the nutrient artery is not rapidly reduced by branching and remains of considerable size until it reaches

the metaphysis (*vide* Fig. 166*a*). Emboli lodge most frequently in the diaphysis of short, hollow bones, as the nutrient arteries branch soon after entering the bone and their lumina are rapidly reduced in

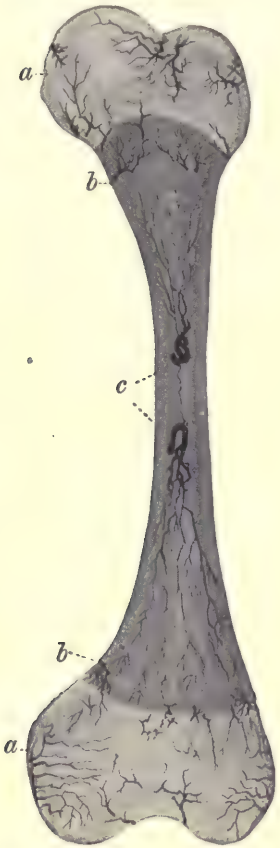

FIG. 166*a*.—FEMUR OF A FOUR WEEKS' OLD CHILD. The periosteum and perichondrium have been dissected away. The intraosseal vessels have been injected with a mixture of mercury and turpentine, and an X-ray picture has been taken. *a*, Epiphyseal arteries; *b*, metaphyseal arteries; *c*, double nutrient arteries.

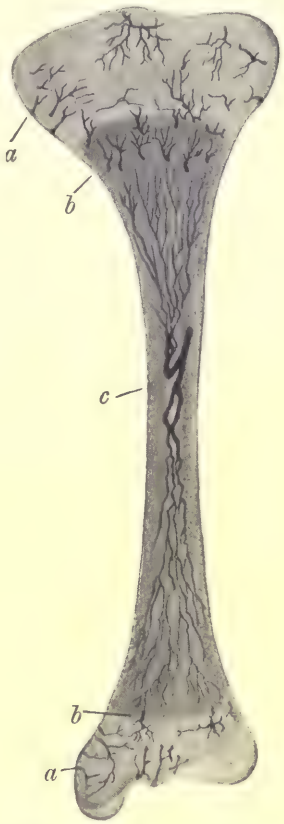

FIG. 166*b*.—TIBIA OF THE SAME CHILD, PREPARED IN THE SAME WAY AS FIG. 166*a*. *a*, Epiphyseal arteries; *b*, metaphyseal arteries; *c*, nutrient artery.

size (Fig. 168) (Lexer). The infarctlike, cuneiform, and conical foci result from the complete occlusion (embolism) of the small end-arteries of the epiphysis and metaphysis of developing bones. A rich anastomosis between these vessels is found only when the cartilages become ossified (Langer). Foci of other shapes are produced by the deposition of small bacterial masses or of infected particles of tissue in the finest vessels or in the capillary

network of the intraosseal vascular system. According to Orth a tuberculous endarteritis involving one of the larger intraosseal vessels may develop and bacilli may be carried from such a focus into the capillaries.

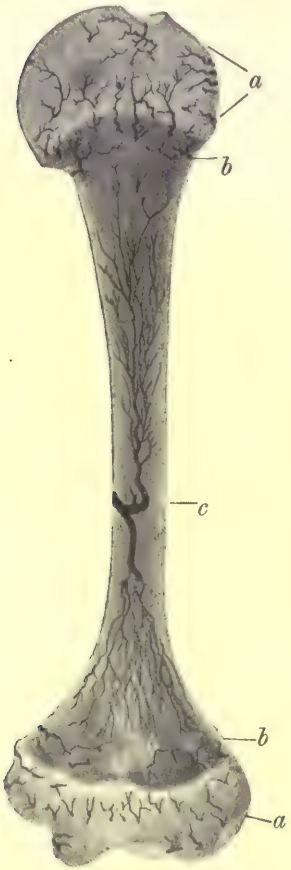

FIG. 167.—HUMERUS OF A NEW-BORN CHILD. Vessels shown as in Fig. 166*a*. *a*, Epiphyseal arteries; *b*, metaphyseal arteries; *c*, nutrient artery.

FIG. 168.—METATARSAL BONE OF THE THUMB OF A NEW-BORN CHILD.

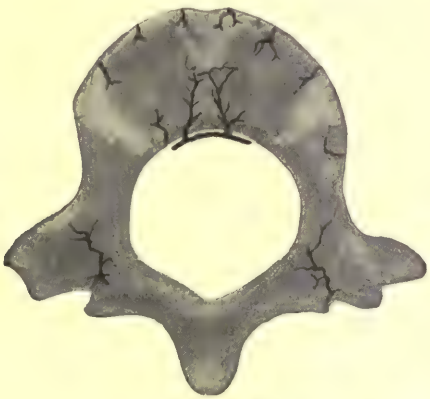

FIG. 169.—THORACIC VERTEBRA OF A FOUR WEEKS' OLD CHILD. The most important arteries pass into the body of the vertebra from behind. Other typical vessels are found in the transverse processes.

The frequent involvement of developing bone by tuberculosis is explained in part by its rich vascular supply. Growing bones are provided with large arteries, giving off many branches, which are directed toward the epiphyseal cartilages, where growth is most active. In adult bone the arteries are small and give off relatively few branches, of which only those in the region of the epiphysis are at all prominent.

Source of the Emboli.—The bacterial emboli causing bone lesions must originate in some primary focus. Tubercle bacilli multiply so

slowly and their growth is so retarded by the lymph nodes that one is not justified in supposing that they may be absorbed directly from recent wounds, uninjured skin and mucous membranes, and enter the circulation. These emboli pass from a diseased lymph node into the thoracic duct or from a focus in the lung into the pulmonary and later into the general circulation.

If symptoms of tuberculosis of bone develop after an injury, it is probable that an encapsulated focus, which up to the time the injury was received had produced no symptoms, has been ruptured. The disease usually follows the lodgment of infected emboli, and the tubercle bacilli are not deposited from the blood stream at the *locus minoris resistentiæ*, as is the case with pyogenic cocci. The results of the experiments made by Friedrich and Honsell show that the deposition of tubercle bacilli is not determined by trauma, but of course an embolus infected with tubercle bacilli may accidentally lodge in a bone at the seat of injury.

Tuberculous osteitis is preëminently a disease of the young, although it may develop at any age. Multiple foci, even in the same bone, are frequent (W. Müller). The disease develops most frequently in the spongy portion of long, hollow bones (epiphysis and metaphysis, or, speaking generally, in the articular ends) and in the diaphysis of short bones.

Pathological Anatomy.—At the point where the tubercle bacilli are deposited, a focus of granulation tissue with miliary tubercles may be demonstrated. In the beginning the focus is grayish red in color and translucent, but becomes yellowish in color as caseation occurs. Wherever such a focus develops, caries results, as the infiltrated bony tissue disappears by lacunar absorption. As a result of the caries, round or tubular cavities (deep defects if the surface of the bone is involved) form, which contain caseous material, resulting from a degeneration of the granulation tissue, and small particles of bone (bone sand). As a rule a tuberculous abscess in bone is circumscribed, rarely becoming larger than a hazel nut, and is separated from the surrounding healthy bone by a connective-tissue capsule (abscess membrane). Small bones, like those of the carpus, may be completely destroyed by caries.

Tuberculous Sequestra.—Usually a sequestrum is formed in the tuberculous focus even when softening, liquefaction, and caseation occur rapidly. A sequestrum is practically always formed in tuberculous foci in children. The trabeculæ, infiltrated with caseous material, become necrotic, although in the beginning they may be thickened as a result of the inflammation. In children, the center of ossification in the epiphysis may become necrotic. Slowly the necrotic bone (sequestrum) is separated from the surrounding parts by a demarcating in-

flammation and the digestive action of the granulation tissue. The tuberculous sequestrum is smooth or somewhat uneven, round or oblong, yellowish white in color, and harder than the surrounding bone, which is softened by granulation tissue. The form and size of the sequestrum correspond approximately to the form and size of the original focus. It may be as large as a pigeon egg. A sequestrum which is not completely separated remains firmly attached at some point or points with the surrounding bone. If both processes, caries and necrosis, are combined, cavities filled with caseous and purulent contents, in which completely separated sequestra lie, are formed.

FIG. 170.—VESSELS IN THE OS INNOMINATUM OF A NEW-BORN CHILD.

The development of a small but appreciable swelling may be the only external sign of a tuberculous focus which has existed for some time. The swelling develops when the focus is situated beneath the periosteum, or when a focus situated within the bone reaches the periosteum or ruptures through it into the soft tissues. Periosteal bone formation (periostitis ossificans) accompanies foci situated in the cortex, especially those foci which are secondary to a tuberculous arthritis. A periostitis accompanies foci situated in the vertebræ, and frequently large defects in the vertebræ are bridged over by newly formed bone. The periosteum produces bone in tuberculosis of the phalanges, metacarpal and metatarsal bones giving rise to the clinical picture of spina ventosa. In rare cases periosteal bone formation accompanies tuberculosis of the diaphyses of larger bones.

Tuberculous foci in the epiphyses of long bones and the resulting sequestra are frequently cuneiform in shape (according to W. Müller in twenty per cent of the cases), the base of the wedge resting upon the articular cartilage, its apex approaching more or less the epiphyseal cartilage. If the latter is destroyed the apex of the wedge may extend into the medullary cavity. The sequestra are the result of an embolic occlusion of the epiphyseal vessels, which branch toward the articular cartilage. In rare cases foci and sequestra of a similar shape are found in the skull (Gangolph) and in some of the short bones (König, Krause). Such foci are frequently found in the pelvis, the base of the wedge

lying in the acetabulum, while the apex is directed above and posteriorly (Fig. 171).

Diffuse Tuberculous Osteitis.—Those cases in which the foci are not surrounded by an abscess membrane or sclerotic bone, but by softened bone (malacia), permeated with miliary tubercles, form the transitional stages to the diffuse tuberculous osteitis. In this infiltrating progressive tuberculosis of bone, described by König, an entire long hollow bone, or the greater part of it (the spongy bone, likewise the medulla and cortex), may be attacked by a caseating and suppurating inflammation. Small or large abscesses then develop in the medullary cavity (osteomyelitis tuberculosa purulenta). This form of tuberculosis of bone is more frequently secondary than primary, being usually secondary to a tuberculous arthritis.

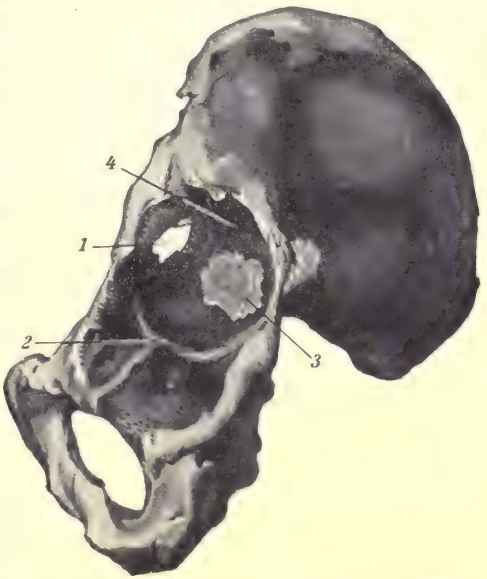

FIG. 171.—TUBERCULOUS CARIES OF THE RIM OF THE ACETABULUM. 1. Perforation inward into the pelvis. 2. Epiphyseal cartilage. 3. Base of the cuneiform sequestrum corresponding to the lower branch of the nutrient artery. 4. Displacement of the acetabular rim upward. Preparation from a child twelve years of age.

Caries Carnosa.—König first described a rare form of tuberculosis of the humerus, which is secondary to tuberculosis of the shoulder joint. The newly formed and old tissues and the bone marrow, permeated with miliary tubercles, are fleshlike and red. This form of tuberculosis has been called caries carnosa.

Spontaneous Healing of Tuberculous Foci.—A tuberculous focus in bone may heal spontaneously. The bacilli are then overcome by the resistance of the tissues, and the focus is either encapsulated or replaced by newly formed connective tissue. As would be expected, spontaneous healing occurs most frequently in small, circumscribed foci in which there is no sequestrum formation. Virulent bacilli may remain in the encapsulated or healed foci, from which recurrences develop when the capsule is destroyed or the resistance of the tissues lowered by trauma. The spontaneous healing of tuberculosis of the spine demonstrates that even the larger sequestra may become encapsulated.

Rupture of a Focus into a Joint or Soft Tissues.—An osteal focus may rupture into a neighboring joint or into the soft tissues. If the focus ruptures into a joint a tuberculous arthritis develops, the clinical

FIG. 172*a*.—TUBERCULOSIS OF THE RIGHT SHOULDER JOINT. Photographed from in front. 1. Very large, completely separated sequestrum. 2. Head of humerus deprived of cartilage and carious. 3. Tuberculum majus. 5. Diaphysis.

FIG. 172*b*.—RESECTED HEAD OF THE HUMERUS. Natural size (after Krause). Preparation made by sawing head of the humerus in frontal plane. 1. Cuneiform sequestrum which extends to the articular surface, the cartilage of which has been destroyed. 2. Extensive caseous infiltration of the head, secondary infection extending from the sequestrum into the tuberculum majus (4) and the diaphysis (5).

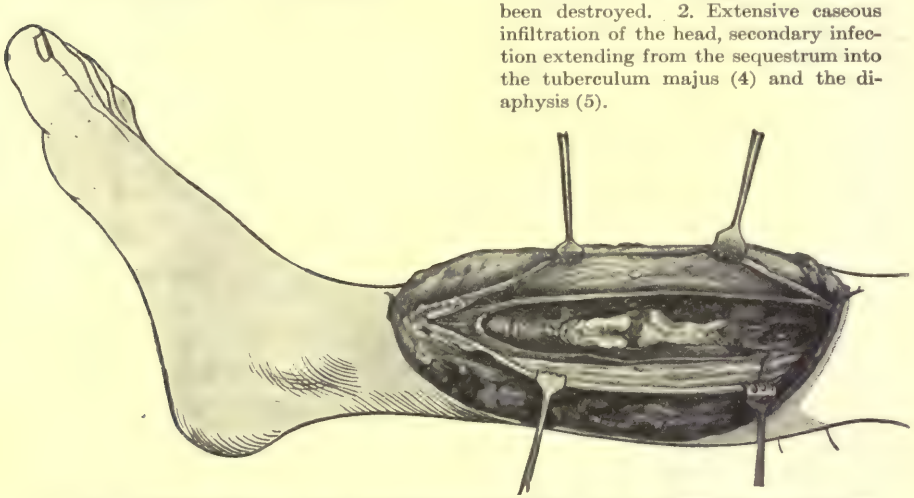

FIG. 173.—TUBERCULOSIS OF THE DIAPHYSIS OF THE TIBIA OF AN EIGHT YEAR OLD GIRL. The foci have been exposed by chiseling away a thick layer of bone. Slight expansion of the bone. Sclerotic bone surrounds the foci.

course of which depends upon the character of the primary focus (König). If there is a tendency to the formation of scar tissue and healing, the tuberculous process extends but little in the soft tissues.

If, on the other hand, there is a tendency to caseation and suppuration, tuberculous or cold abscesses form. These abscesses (gravitation abscesses) follow the force of gravity, but may burrow against it. Large and very extensive cold abscesses may develop from exceedingly small foci. When the foci lie directly beneath the skin, chronic fistulæ and ulcers form after the rupture of the abscess.

Clinical Course.—The clinical picture of tuberculosis in different bones varies. The articular ends of long hollow bones are most frequently involved. Round or cuneiform foci (foci of granulation tissue and pus with or without sequestra) form which may rupture into the joint or into the para-articular soft tissues; foci in the cortex, beneath the periosteum, or within the medulla of the diaphysis are much less frequent. Diffuse tuberculosis of the epiphysis and diaphysis of large bones is rare and most frequently follows a severe form of tuberculosis arthritis. In the short, hollow bones, the changes are most pronounced in the diaphysis. The cortical layer of bone is destroyed from within, and as the process reaches the surface the periosteum is stimulated to the production of

FIG. 174.—TUBERCULOUS OSTEITIS OF THE FIRST AND SECOND PHALANGES OF THE INDEX FINGER WITH ABSCESS FORMATION. Subcutaneous abscesses are also present upon the dorsum of the hand.

FIG. 175.—TUBERCULOUS OSTEITIS OF THE PROXIMAL PHALANX OF THE INDEX FINGER. Flask-like expansion of the finger caused by some expansion of the bone and the development of masses of granulation tissue.

new bone, replacing partially the cortical bone, which is being destroyed. The entire diaphysis may be filled with caseous material or may contain one large sequestrum. The old name *spina ventosa* is frequently applied to tuberculosis of the phalanges, metacarpal, and metatarsal bones. Tuberculous osteitis of the phalanges gives to the fingers a peculiar, bottle-shaped form, which may also be produced by a tuberculous periostitis.

Tuberculosis of the vertebræ is very common; they are the most frequently attacked of all the short bones. Frequently multiple foci develop upon the anterior surface of the body of a vertebra beneath the anterior longitudinal ligament or within the bone. These foci soften, and the diseased bone is then crushed by the weight of the superimposed vertebræ. The spine of the diseased vertebra becomes prominent, and an angular kyphosis or gibbus develops. If a number of vertebræ are diseased, the kyphosis will be rounded instead of angular. Spontaneous healing of inaccessible foci occurs frequently. The well-known deformity "humpback" is the result of a healing of a tuberculous spondylitis in malposition. As the tuberculosis heals there is a periosteal formation of bone upon the anterior surface of the vertebræ. Gravitation abscesses frequently develop in the course of tuberculous spondylitis. The retropharyngeal and psoas abscesses are the best-known examples. Tuberculosis of the laminæ is much rarer than that of the bodies of the vertebræ (being most frequent in the atlas and axis).

Fig. 176.—Tuberculous Caries of the Twelfth Thoracic, First and Second Lumbar Vertebræ; Marked Formation of Osteophytes upon the Anterior Surface of the Sacrum. Left half of the pelvis removed.

Tuberculosis of the carpal and tarsal bones is frequently followed by severe tuberculous arthritis.

The ribs are most frequently involved of any of the flat bones. Superficial, subperiosteal foci, which may become very extensive, and larger osteal foci with sequestra develop. The frontal and parietal are the skull bones most frequently attacked. The foci of granulation tissue and sequestra may perforate the bone and extend to the dura; the dura will be exposed when an abscess of the scalp is incised, and the granulation tissue and sequestra removed. Tuberculosis develops frequently in the outer part of the orbital process of the superior maxilla and in the malar bone, where it articulates with the latter. It may also develop in the scapula, clavicle, sternum, and ilium.

Symptoms.—Clinically, as a rule, the first symptoms of tuberculosis of bone consist of pain and swelling. These develop as soon as the focus within the bone reaches the periosteum and soft tissue. Suppuration in the soft tissues and rupture into a joint give rise to definite symptoms (see below). The tuberculous abscess in the soft tissues develops slowly and the skin covering it does not present any of the signs of inflammation (cold abscess). There is but a slight elevation of temperature in uncomplicated tuberculosis of bone; frequently there is no fever at all. High fever indicates a secondary infection with pyogenic bacteria, which frequently occurs when a suppurating fistula communicates with the abscess cavity, or it indicates the beginning of a miliary tuberculosis.

Diagnosis.—Usually the diagnosis of tuberculosis of bone is not difficult. Tuberculosis of the vertebræ, skull and facial bones, phalanges, and a number of other bones presents a very definite clinical picture. The slow development of the abscess following the inflammation of bone, the absence of local as well as general symptoms of acute inflammation, the distention of the skin, the rupture of the abscess with subsequent development of chronic fistulæ and ulcers with flabby, yellow granulations and undermined edges, and finally the swelling of adjacent lymph nodes leave no doubt as to the nature of the pathological process. Besides, tuberculosis develops most frequently in the bones of weak individuals, in whom there are already evidences of some other form of tuberculosis, such as tuberculosis of the lungs, lymph nodes, joints, skin, or mucous membrane.

Sometimes it is difficult to make a differential diagnosis between tuberculous and suppurative osteomyelitis, especially when the foci produced by the latter are small, are situated in the articular ends of the bone, and pursue a subacute or chronic course. Expansion of the bone speaks against tuberculosis. Only in the rare cases of primary tuberculosis of the shaft of long bones does the cortical layer of bone become

expanded to resemble clinically spina ventosa, already described in tuberculosis of short, hollow bones. If there are no characteristic abscesses, fistulæ, or ulcers, it may be impossible to make an accurate differential diagnosis before operation. The caseous pus and the round, small sequestra of tuberculosis are very different from the thick, creamy pus, and the irregular, notched sequestra resulting from pyogenic infection. In doubtful cases the demonstration of cocci in the pus, or of tubercle bacilli in the granulation tissue determines the diagnosis. The diagnosis of tuberculosis may sometimes be made with the X-ray if cuneiform sequestra are present.

Treatment.—The most important indication in the treatment of tuberculosis of bone is to remove accessible foci, especially if they suppurate. This can be done when the epiphyseal focus has ruptured externally, when the inflammation involves the bones of the skull and face, ribs, etc. Large epiphyseal foci should be operated upon as soon as possible in order to protect the joint from the disease. The operation should also be performed as early as possible in tuberculosis of the short, hollow bones. If the disease is allowed to progress, the form of the fingers and toes is destroyed and changes occur which interfere with their function. The early operation should be performed whenever the focus can be demonstrated and is accessible.

All operations upon the extremities should be performed under Esmarch's artificial ischæmia. Frequently removal of the focus with a sharp spoon, after an incision has been made down to the bone and the periosteum has been reflected to either side, is sufficient. In other cases it will be necessary to use a chisel to remove a sequestrum which is not completely separated, or to expose and remove the contents of the medullary cavity, as in the treatment of suppurating osteomyelitis. If the latter is necessary, the epiphyseal cartilages should always be spared when possible (Fig. 173). The resulting cavities should be tamponed with iodoform gauze or filled with iodoform-glycerin emulsion after the skin sutures have been inserted. Resection of the diseased parts of flat bones (ribs, scapula, and ilium) gives the best results. Resection of the joint is indicated if the focus in the bone has ruptured into the joint and produced severe destructive lesions; amputation is occasionally indicated in the infiltrating, progressive form of tuberculous osteitis. The treatment depends upon the bone involved (e. g., vertebra).

[In the treatment of all forms of tuberculosis great stress should be laid upon the necessity of improving the general condition and raising the resistance of the patient. Out-of-door life, good food, plenty of sunshine, and rest are as essential as surgical treatment.]

The treatment of tuberculous abscesses developing from osteal foci differs, depending upon the position of the latter.

In small abscesses with a subjacent, osteal focus, the following treatment is to be recommended: incision, removal of the abscess membrane, and insertion of a tampon of iodoform gauze. If the abscess is very large, a tampon should not be inserted, but the incision should be sutured and iodoform-glycerin emulsion then injected between the stitches into the cavity (Billroth). This treatment cannot be employed when the abscess has opened spontaneously or is acutely inflamed, for in those cases the dangers associated with a phlegmon require free incision and open treatment.

It is difficult to cure abscesses by incision when there is an inaccessible osteal focus. A psoas abscess is a good example of a lesion of this character. In these cases the osteal focus keeps up a continuous secretion, and after evacuation of the contents of the abscess a fistula forms which it is often difficult to protect from secondary pyogenic infections. These secondary infections aggravate both the local and general condition, and therefore incision of gravitation abscesses should be attempted only after the treatment by aspiration has been tried without success.

Large syringes and canulæ should be used in aspirating cold abscesses, for the pus contains numerous fragments of tissue and flakes of fibrin. If these occlude the canula an attempt should be made to remove them with a wire or fine probe. The canula should be inserted obliquely through the skin and soft tissues, so that the edges of the wound will agglutinate rapidly when the canula is removed. The formation of a fistula may be prevented in this way. For the same reason it is recommended that a small incision be made before the insertion of very large canulæ (Henle).

Iodoform-glycerin emulsion should be injected after the removal of the pus (von Mosetig-Moorhof, Billroth, von Mikulicz). The results following the use of this emulsion are better than those following the use of iodoform-ether, carbolic acid, and zinc chlorid solutions, and it is generally employed. A ten per cent emulsion of iodoform in glycerin is employed. The emulsion should be thoroughly shaken before being used, and should be made fresh frequently (at least once a week). According to the experience of von Bergmann's clinic, it is not necessary to sterilize this emulsion. The activity of the emulsion seems to be reduced by sterilization, and besides iodin, which is harmful, is set free in the process.

This emulsion may be injected into a large abscess cavity through the canula used for aspirating the pus. Fifty c.c. (in small children 10 c.c.) may be injected into such a cavity. If the abscess membrane has been removed and there are raw surfaces, only from 10 to 20 c.c. (in children a corresponding smaller amount) should be injected, unless

free escape is provided between the stitch-holes, because of the dangers of poisoning following absorption.

The injections should be repeated after intervals of from two to four weeks. Frequently abscesses heal under this treatment. The emulsion also has a favorable action upon the osteal focus. Careful asepsis should be practiced while the injections are being made in order to prevent secondary infection. If the latter occurs the abscess should be incised immediately and treated by the open method. Fistulæ following the rupture of gravitation abscesses frequently heal when iodoform-glycerin emulsion is injected and the granulation tissue lining the fistula is repeatedly curetted away.

The value of the emulsion depends upon the irritation produced by the iodoform which remains in contact with the tissues for some time. As a result of this irritation a healthy granulation tissue, which tends to contract and in which new tubercles cannot develop, forms, while the old tuberculous granulation tissue is destroyed. Iodoform, which is decomposed in the tissues, undoubtedly has some influence upon the bacteria, but this is little.

A word of warning should be spoken against the use of excessive amounts of the emulsion. Severe, even fatal, iodoform intoxication has been observed after the injection of the emulsion into abscess cavities and joints. Many patients are very susceptible to iodoform, and the use of even small amounts of the emulsion may be followed by a general reaction associated with high fever and the symptoms of intoxication. Naturally the emulsion should not be used when the patient gives a history of susceptibility to iodoform. Sometimes after the injection there are an increased pulse rate and an elevation of temperature associated with an acute, transitory nephritis with hæmoglobinuria, which has been regarded as due to the glycerin (Henle).

(g) TUBERCULOSIS OF JOINTS (TUBERCULOUS ARTHRITIS)

Tuberculosis of joints follows hæmatogenous infections, the rupture of a primary osteal focus into a joint; more rarely, a tuberculosis of adjacent tendon-sheaths. It is possible for infection to be carried through lymphatic vessels from diseased lymphatic nodes situated near a joint. Tuberculous arthritis is rarely a primary infection, as there is a focus in some other part—for example, in the lung, bronchial or mesenteric lymph nodes, mucous membrane or skin (König). Trauma has the same relation as an etiological factor to tuberculous arthritis that it has to tuberculous osteitis.

In the majority of cases of tuberculous arthritis the disease begins in the articular end of one of the bones entering into the formation of the joint, and the synovial membrane becomes involved secondarily.

Primary synovial tuberculosis is, however, more frequent than was formerly considered to be the case. [Müller's statistics, published from König's clinic, show that in 232 cases of tuberculous arthritis, the disease began in bone in 158, in the synovial membrane in 46, and that in 28 cases the origin could not be determined.] According to König a number of the osteal foci must be regarded as secondary to a synovial tuberculosis.

The disease develops most frequently in the first two decades of life. The knee, hip, and elbow joints are most frequently involved, and in the order of frequency as given. The joints may be involved in acute general miliary tuberculosis.

Pathological Anatomy of Different Forms.—The formation of tuberculous granulation tissue, in which are imbedded numerous miliary tubercles, a chronic reactive inflammation of the synovial membrane, and the production of an exudate follow the deposition and multiplication of the tubercle bacilli in the synovial membrane of the joint. It makes no difference whether the bacilli are carried into the synovial membrane by the blood or whether they pass into the membrane after a primary osteal focus has ruptured into the cavity of the joint. The clinical course of the disease is determined by the character of the granulation tissue and the exudate, and by the secondary changes in the cartilages and bone.

The tuberculous granulation tissue, which appears first upon the synovial membrane and later extends to the articular cartilages at the line of attachment of the membrane, may tend to cicatrize (the fibrous, dry, granulating form) or to degenerate and disintegrate (soft, sloughing form). In the first form the synovial membrane, in the inner layers of which are found many tubercles, is considerably thickened and its free surface is partially or completely covered with pale, grayish red tuberculous granulation tissue which only occasionally caseates or suppurates. In the beginning of the disease there is generally a serous or a serofibrinous exudate.

In the caseating form of tuberculous arthritis the synovial membrane is covered with and partly transformed into soft, spongy granulation tissue, while the para-articular tissues are œdematous. Circumscribed caseous foci and abscesses frequently form within this granulation tissue, which may rupture into the cavity of the joint, and a tuberculous suppuration, such as frequently follows the rupture of primary osteal foci into the joint, develops in this way. If the process gradually extends through the synovial membrane, foci of granulation tissues and abscesses develop in the para-synovial tissues, which may later rupture through the skin and lead to the formation of fistulæ.

Suppurative Tuberculous Synovitis.—Another and rare form of suppurative tuberculous arthritis, which has been observed especially in children (in the knee and hip joint), develops rapidly after the miliary tubercles form. The synovial membrane, which is but little thickened, contains considerable numbers of miliary tubercles, and its surface is covered with an abscess membrane which can be easily removed (Synovitis suppurativa tuberculosa, König).

Nodular Form of Tuberculous Arthritis and Villous Arthritis.—Often circumscribed masses of granulation tissue, accompanied by a serofibrinous exudate, develop within the joints. The masses, which may become as large as a pigeon egg, are grayish red in color and are attached to the fibrous layer of the synovial membrane by a pedicle (nodular form of tuberculous arthritis, tuberculous fibroma, König). These masses show no particular tendency to caseate and contain but few tubercle bacilli. In other cases villous-like growths, which branch like a tree, develop in the synovial membrane and thick, fibrinous masses are deposited upon the membrane or in the joint as the result of the chronic inflammation (villous tuberculous arthritis) (Fig. 177).

Fig. 177.—Proliferation of the Synovial Villi in Tuberculosis of the Knee Joint. (After König.)

An attempt has been made to explain the development of the rare lipoma arborescens (Johannes Müller), which is most frequently associated with synovial tuberculosis of the knee joint, as due to the growth of the synovial villi resulting from the chronic inflammation. Similar growths are found in other diseases of the joints, such as chronic rheumatism, arthritis deformans, and syphilis. Of course tuberculosis may develop in a joint which already contains a lipoma arborescens (Krause).

The contents of the joint in the beginning of the disease or in mild forms of the disease are serous in character (hydrops articularis tuberculosus serosus, König), the fluid being yellowish and clear or somewhat clouded. White flakes in the exudate indicate the presence of fibrin (hydrops fibrinosus, König). Fibrinous masses, which may form a soft, white membrane, may be found in the joint cavity, most frequently in the recesses of the joint and along the line of reflection of the capsule. Frequently these fibrinous masses lead to the formation of villouslike structures and free-bodies.

Rice Bodies.—The so-called rice bodies, *corpora oryzoidea*, which may fill the greater part of the diseased joint, are round and compressed, resembling seed-corn in shape. They are soft and white and are covered with an exceedingly slippery exudate. Often similar structures are attached to the synovial membrane by a pedicle; frequently they are free in the joint, but a connective-tissue center indicates that the pedicle has been destroyed and that the bodies which are free in the joint were formerly attached. These bodies should always be regarded as the product of tuberculous inflammation, for they, like the exudate, contain tubercle bacilli; it may be only a few, but when injected into animals they produce tuberculosis.

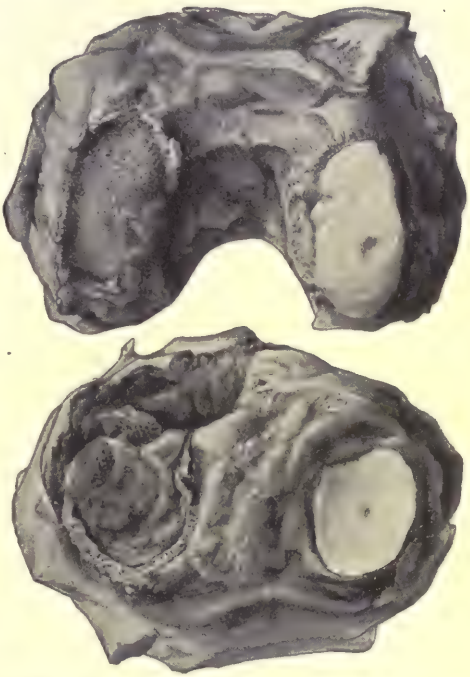

FIG. 178.—TUBERCULOSIS OF THE KNEE JOINT (RESECTION PREPARATION). The articular cartilages covering the corresponding surfaces of the external condyles of the femur and tibia are destroyed. Small depressions may be seen in the articular cartilage covering the external condyle which is still retained. The edges of the cartilage are infiltrated with tuberculous granulation tissue. Between the cartilage and the defect fungous masses may be seen.

Origin of Rice Bodies.—According to König, Landow, and Riese the pedunculated and free rice bodies develop from deposits of fibrin; according to Schuchardt, Garrè, and Goldman they are to be regarded as the degeneration products of diseased synovial membrane (fibrinoid necrosis). The separation of the fibrinous or degenerated layers of the synovial membrane and

the form of the free-bodies are probably due to the movements of the joint.

A purulent tuberculous exudate is rare, and is found only in the severe forms of joint tuberculosis.

The destruction which the joint undergoes is not limited to the ligaments and articular fibro-cartilages, which are infiltrated with tuberculous granulation tissues, but also extends to the articular cartilages and the subjacent bone.

The articular cartilage is never the seat of primary tuberculosis, although it may be attacked and destroyed when primary osteal or articular foci extend to it. Tuberculous granulation tissue extends from the synovial membrane to the articular cartilage and produces in it small holes, funnel-shaped depressions, and large defects which may extend down to the bone. According to König, these changes are due in the first place to the action of the organized fibrinous masses. The same thing is observed in hæmophiliac joints. The destruction of the articular cartilage may follow the development of an osteal focus, the base of a sequestrum projecting into the joint and being worn off by the movement of the latter.

Fig. 179.—Coronal Section of the Lower End of a Femur, which was Amputated because of Extensive Tuberculosis of the Knee Joint. The spongy tissue of the epiphysis has been transformed into simple granulation tissue without tubercles (osteitis granulosa). The articular cartilages are raised from the bone by this tissue.

Another specific process observed in primary tuberculous synovitis is the transformation of the yellow marrow of the spongy tissue of the epiphysis into simple granulation tissue without tubercles (osteitis granulosa, Fig. 179). As the result of the extension of the inflammation the bony trabeculæ undergo lacunar resorption and are destroyed by osteoclasts and the bone becomes soft and porous. Masses of granulation tissue extend into and through the articular cartilage and project as fungous growths into the joint cavity. The articular cartilage is perforated like a sieve (von Volkmann). In other cases the granulation tissue separates the articular cartilage from the bone. The articular cartilage then appears as a hump upon the bone; in the head of the femur as a hood. Later the thinned cartilage is broken

down or becomes stratified (Fig. 180). Tubercles develop only when the granulation tissue of this form of osteitis extends into the joint. The tissue may then undergo caseation and puriform softening; the bone, necrosis and caries (joint caries).

The articular cartilages and bone are destroyed by suppuration or by the pressure of masses of tuberculous granulation tissue.

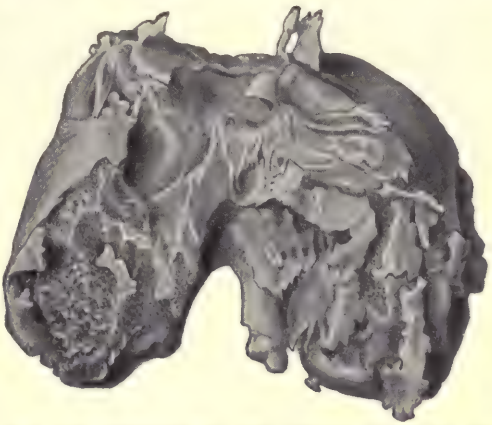

Fig. 180.—Section of the Femur Involved in Tuberculosis of the Knee Joint. The articular cartilage is raised and separated from the bone; the roughened, granulating bony surface may be seen below it. (After König).

Caries Sicca.—There is a special form of tuberculous arthritis (caries sicca, von Volkmann) in which the secondary changes in the articular cartilages and bone develop without exudation. In this form of arthritis a thin layer of tuberculous granulation tissue, which tends to cicatrize, forms and slowly destroys the cartilage and bone. An entire epiphysis may be destroyed while the granulation tissue is transformed into cicatricial masses. This form of tuberculosis is seen most frequently in the shoulder and hip joints.

Reactive Changes.—The reactive changes occurring in the surrounding tissues also belong to the anatomical picture of joint tuberculosis. These changes affect the connective tissues and periosteum. All the soft tissues, the joint capsule, the ligaments, tendon-sheaths, likewise the subcutaneous connective tissue, are transformed by the chronic hyperplastic inflammation into firm cicatricial masses which, because of the atrophy of the fat and accompanying œdema, often acquire lardaceous and gelatinous characteristics.

The same inflammatory irritation, after persisting for a long time, produces changes in the bone. Especially in the suppurative form a large number of osteophytes may develop about the joint involved as a result of an ossifying periostitis.

Spontaneous Healing.—Tuberculous granulations may cicatrize and become transformed into scar tissue in which the tubercles are destroyed or encapsulated. Larger foci of granulation tissue and caseous masses may also be encapsulated by this tissue. The peri-articular tissues may also contract and the movement of the joint become limited as the result of healing or apparent healing. This spontaneous healing

is of great importance from a therapeutic viewpoint, and treatment should be instituted which favors it.

If the cartilage and bone are but partially destroyed, if the infiltration of the bone is not extensive, and if there is no suppuration, spontaneous healing of the tuberculous process may occur. Adhesions which may become ossified then develop between the opposing surfaces, and fibrous or bony anchylosis develops. Concerning anomalies in position following healing, *vide* below.

Clinical Course and Symptoms.—The symptoms and the sequelæ of the various forms of tuberculous arthritis differ, and the clinical course of the disease has protean characteristics, but the symptoms of tuberculosis of some joints are pronounced and characteristic.

Usually the onset of tuberculous arthritis is gradual and the course chronic. According to Rovsing, in small children, especially in nurslings, suppurative tuberculous arthritis begins acutely with high fever. This form of the disease develops most frequently in the knee joint.

The prodromata of tuberculous arthritis are weakness and a proneness to fatigue of the extremity involved. If there is an osteal focus the patient will have noticed for a long time a radiating pain, which finally locates in the joint when the arthritis develops. Frequently exertion or slight trauma, such as movements of or bearing weight upon the joint, or pressure upon the head of the femur if the hip is involved, produces an exacerbation of the disease accompanied by some fever and severe pain. The first objective symptoms are a moderate amount of swelling due to exudation into the joint, thickening of the capsule, œdema of the para-articular tissues, and fixation of the extremity in characteristic positions (coxitis, abduction and outward rotation, gonitis, flexion, etc.).

In the beginning the diseased joint is fixed to prevent pain, which follows every movement. The patient attempts to hold the diseased joint in the position which is the least painful (König). This is especially pronounced in the lower extremity, when the patient continues to walk after the disease has developed. The position assumed is that in which the capacity of the joint is greatest (Bonnet's experiments upon cadavers), and is partly the result of reflex muscular contraction. In the beginning of the diseases the abnormal position may be easily corrected under anæsthesia.

König distinguishes the following forms of tuberculous arthritis: tuberculous hydrops, granulating tuberculous arthritis (fungus articuli, tumor albus), suppurative tuberculous arthritis.

1. *Tuberculous Hydrops.*—This form of the disease is seen most frequently in adults. It develops in the knee joint, but also in the ankle and elbow joints. The symptoms usually develop gradually and there

is no particular reaction, although at times the onset is acute. The joint becomes filled with a serous exudate, the capsule becomes distended, and fluctuation may be elicited. As a rule, there are no other distinct evidences of tuberculosis (*vide* Fig. 181). The movements of the joint are restricted as the articular structures are distended and painful. Contractures rarely develop.

Fig. 181.—Tuberculous Hydrops of the Right Knee.

Flakes of fibrin in the fluid removed by aspiration often indicate the tuberculous nature of the process. A definite diagnosis can be made, if there are no other symptoms of tuberculosis, only by a microscopical demonstration of the bacilli or the positive results following inoculation of animals with the fluid. It is often difficult to differentiate between this form of arthritis and the arthritis due to trauma, floating bodies, syphilis, chronic gonorrhea, and that accompanying suppurative osteomyelitis.

The exudate may gradually disappear and spontaneous healing may occur. Recurrences are frequent. Very often the serous exudate is merely the first stage in the development of a fungous tuberculous arthritis, the exudate gradually disappearing and fungous masses developing. The diagnosis is not so difficult when there are large amounts of fibrin in the exudate (hydrops fibrinosus), for then peculiar grating sensations may be elicited when the soft swellings in the capsule, which fluctuate but little, are palpated. These grating sensations are caused by the displacement of fibrinous masses, villi and rice bodies upon each other. Similar deposits and villous growths may be present in other diseases of joints (hæmophiliac joints, chronic rheumatism, arthritis deformans), and for this reason the tuberculous nature of the process may long be concealed.

2. The *granulating form of tuberculous arthritis* (fungus) is the most frequent. Joints which are superficial gradually assume a characteristic shape, when the granulation tissue develops in the joint and the para-synovial tissues become swollen. Such joints become spindle-

shaped, as their outlines are destroyed by the distention of the capsule and the swelling of the para-articular tissues. This becomes more pronounced as the disease progresses, for the muscles above and below the swelling become atrophic (Figs. 182–184).

The soft masses of granulation tissue often impart to the palpating finger the sensation of pseudo-fluctuation. Often the swelling is hard

Fig. 182.—Tuberculosis of the Left Knee (Granulating Form, Fungus).

and resistant, as the para-synovial tissues have been transformed into cicatricial tissue and the skin adherent to the indurated tissues is tense, shining, and anæmic (tumor albus). If the granulation tissue tends to cicatrize, complete healing may occur, but the joint will be anchylosed, and, if not properly treated, in malposition. The contractures developing when proper treatment is not instituted are due to the shortening of the muscles, which are no longer used, and to cicatricial contraction of the capsule and of the surrounding tissues. If the granula-

tion tissue caseates and suppurates, abscesses and fistulæ develop and there is an evening rise of temperature.

The greater the destruction of the joint the more marked the contractures, as the articular ends of the bones, after having been destroyed, become displaced. For example, dislocation of the hip follows destruction of the head of the femur or of the upper and posterior part of the acetabulum (Fig. 171), genu, valgum and varum destruction of the bones

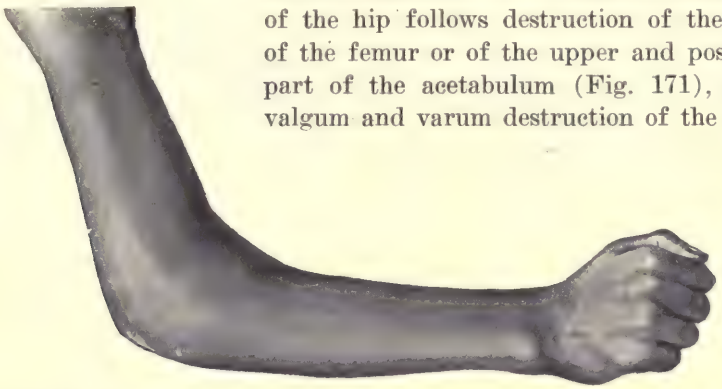

Fig. 183.—Tuberculosis of the Elbow Joint (Granulating Form with Abscess Formation).

forming the knee joint. Pathological dislocations and subluxations may follow the distention and weakening of the ligaments by the granulation tissue and exudate as well as the destruction of the bones. Volkmann has called the former distention-, the latter destruction-dislocations. They may develop gradually or after some insignificant injury.

Diagnosis.—A beginning fungous tuberculosis of a joint may be most easily confused with an osteal sarcoma (periosteal as well as myeloid) which develops into a joint. Sometimes it is necessary to watch the case for some time before a diagnosis can be made; often, Roentgen-ray pictures and exploratory incision are necessary. Arthritis occurring in hæmophilia and associated with periosteal gumma resembles clinically this fungous form of tuberculous arthritis.

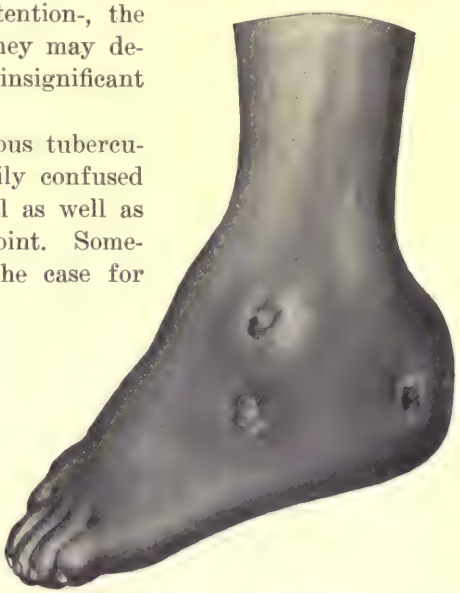

Fig. 184.—Tuberculosis of the Ankle Joint (Granulating Form with Fistulæ).

3. *Suppurative tuberculous arthritis* (cold abscess of joints) is much rarer than the preceding

form. It is usually secondary to a primary synovial tuberculosis, pursues a chronic course, and is accompanied by an exudate into the joint. Severe pain and fever are absent, and, as a rule, a diagnosis

FIG. 185.—HEALED TUBERCULOSIS OF THE KNEE JOINT. Bony anchylosis with backward displacement of the tibia.

of hydrops is made, especially if the knee joint is involved. Contractures frequently do not develop. If the disease is accompanied by fever, and if abscesses develop in the soft tissues, the diagnosis is no longer difficult.

Prognosis.—The prognosis of tuberculous arthritis depends upon the general condition of the patient and upon whether or not there are foci in other organs. A majority of these cases die of tuberculosis of one of the viscera, of exhaustion, or amyloid degeneration. In other cases patients die of an acute general miliary tuberculosis or of a general infection following a suppurative or putrefactive inflammation which extends along fistulæ to the diseased joint.

The prognosis is better in the cicatrizing than in the suppurating forms of tuberculous arthritis. In Bruns's statistics 77 per cent of the non-suppurating forms of tuberculous coxitis healed, 23 per cent ended fatally, while in the suppurating form only 42 per cent recovered and 52 per cent ended fatally.

There is also some difference as regards prognosis depending upon the age of the patient, it being considerably more favorable before than after the fifteenth year. Only in rare cases is the function of the joint completely restored. This occurs most frequently in tuberculous hydrops. Some limitation of motion is the rule; complete anchylosis is frequent.

Recovery, even with function, is frequently only apparent, for tuberculous tissue, from which recurrences may develop, remains encapsulated.

Treatment.—Many forms of tuberculous arthritis tend to heal *spontaneously*, and therefore the first treatment which is instituted should be conservative. If conservative treatment is unsuccessful, an operation (which is usually required only in severe cases or in those cases

in which the general condition of the patient is poor) is indicated. In the conservative treatment an attempt should be made to promote the healing of the tuberculous process with preservation of the structures entering into the joint, if possible, with motion. If this is impossible, an attempt should be made to promote healing with the parts in the most useful position.

A correctly applied plaster-of-Paris bandage is extremely valuable in the treatment of tuberculous arthritis. It places the joint at absolute rest, protects it from injury, and hastens by even and mild compression the absorption of the exudate. If contractures are present they should gradually be corrected by extension with weight and pulley.

Rest in bed is absolutely indispensable if the joints of the lower extremity are involved, as any movement or pressure injures the diseased joint. It should be maintained as long as the joint is painful to pressure or when weight is borne upon it and there is an elevation of temperature. The plaster-of-Paris bandages should be applied after the contracture has been overcome by extension with weight and pulley. This bandage should be changed after six or eight weeks, at which time the skin should be washed and a powder or salve applied in order to prevent eczema. If there are fistulæ, fenestra should be cut in the cast in order that the dressings may be changed frequently.

If the painful stage has passed (frequently it requires months) and the swelling has subsided, the proper dressing or apparatus may be applied and the patient allowed to get up. The dressing or apparatus used should be so applied that the joint is kept at absolute rest, is in the proper position, and bears no weight. [A proper apparatus may be procured from different instrument makers. A special surgery should be consulted for the different apparatus used in the treatment of tuberculosis of the various joints.] A simple plaster-of-Paris dressing which includes the entire extremity and pelvis is very satisfactory in the treatment of lesions of the lower extremity. The bandage should fit the pelvis, especially the ischial tuberosities, well, as the entire weight will be transmitted to them.

Later, when the joint is able to bear some weight, a light dressing made of plaster-of-Paris, water-glass, leather, or felt, which can be removed at night, should be worn to prevent contractures. These may be discarded when there is no longer any tendency to the development of contractures.

The contractures due to reflex muscular contraction and cicatricial contraction of the capsule, which are almost always present when the patients present themselves for treatment, may be overcome in a few days by extension with weight and pulley. If ambulatory treatment

is indispensable and a plaster-of-Paris cast must be applied, the contracture may be gently corrected under anæsthesia. If the contracture is due to the cicatricial contraction of the para-articular tissues and shortening of the muscles, gradual extension with the weight and pulley is the best procedure, as forcible correction (*brisement forcé*) may rupture encapsulated foci and cause a local exacerbation or a general miliary tuberculosis. If it is absolutely necessary that ambulatory treatment be instituted, the correction should be made at a number of different sittings, the correction obtained at each sitting being maintained by the application of a well-fitting plaster-of-Paris dressing.

Frequently an operation (resection of the joint, osteotomy) is required to correct the deformities resulting from fibrous and bony anchylosis.

The intra-articular medicinal treatment, introduced by Hüter, Billroth, and von Bruns, is of great value in the conservative treatment of these cases. Of the many agents which have been tried, ten per cent iodoform-glycerin emulsion, employed first by von Bruns and others, is the most useful. (For details concerning iodoform-glycerin emulsion, *vide* p. 427).

Injection of Iodoform-Glycerin Emulsion.—After the removal of the exudate 10 to 20 c.c. of the emulsion, in children 5 to 10 c.c., should be injected into the large joints. The joint should be gently rubbed or kneaded after the injection in order to distribute the emulsion. In fungous tuberculosis small amounts should be injected at a number of different points, the injections being repeated after intervals of from two to four weeks, and the exudate which has reformed being removed if necessary. Between injections the joint should be immobilized in a plaster-of-Paris cast as described above. Of course the asepsis must be as nearly perfect as possible when these injections are made.

Para-articular abscesses and fistulæ should also be treated by injections (*vide* Tuberculosis of Bone).

A two to three per cent solution of carbolic acid may be used in patients who are very susceptible to iodoform.

The conservative treatment (immobilization), which is frequently combined with injections of iodoform-glycerin emulsion, is indicated in every recent case of tuberculous arthritis and should be tried for some time in old cases. If the local or general condition does not improve, if the tuberculous process is extending, if there is a large primary osteal focus or severe secondary involvement of cartilage and bone, operative treatment should no longer be delayed.

Conservative treatment is not suited for the cases in which there is a tuberculosis of some of the viscera (lung or kidney), in which the general condition is not good (old people), and in which the fistulæ

communicating with the joints have become infected with pyogenic or putrefactive bacteria.

Usually, conservative treatment must be continued for a number of years, as recurrences are frequent and the process of repair is prolonged.

According to König, the results of conservative treatment are more favorable in those cases developing before the fifteenth year than later. His statistics concerning the results of the conservative treatment of tuberculosis of the knee joint are as follows: Before the fifteenth year, 52 per cent recover; after it, 22 per cent. Henle believes that 79.3 per cent of the cases of tuberculosis of the large joints recover before the fifteenth year; 62.5 per cent after it.

Bier has observed that patients with passive congestion of the lungs (e. g., in heart lesions) acquire a certain immunity against tuberculosis, and has advised and used extensively passive hyperæmia in the treatment of tuberculosis of bones and joints. Helferich had formerly employed passive hyperæmia in the treatment of delayed union following fractures, and it had been employed earlier by Dumreicher in the treatment of old ununited fractures. In producing the hyperæmia an elastic constrictor should be applied above the diseased joint. It should exert just enough pressure to induce a warm passive hyperæmia. If the extremity becomes blue and cold, the constrictor should be loosened or removed and reapplied. Surgeons differ as to the value of passive hyperæmia in the treatment of tuberculosis of bones and joints.

Operative treatment is indicated in those cases in which there is tuberculosis of some of the viscera (lung and kidney), in those in which there is extensive bone involvement, and in those cases which do not improve under conservative treatment. The operation should be performed under artificial ischæmia, and incisions should be employed which give the best view of the parts involved. All diseased or apparently diseased tissue should be removed with knife, scissors, and sharp spoon. When the diseased synovial membrane is entirely removed, the bone should be examined and any foci which are found should be removed. Para-articular abscesses and foci of granulation tissue should be opened; their walls should be excised or removed with a sharp spoon. In simple cases this partial arthrectomy is sufficient. If the articular cartilages and the epiphyses are destroyed, a typical resection must be performed. In young subjects the epiphyseal cartilage should be preserved in order to prevent later shortening of the bone.

The wound should not be completely closed; any recesses should be tamponed or drained. Healing, with fibrous anchylosis with some movement or complete bony anchylosis, occurs in about two months. A light splint, a plaster-of-Paris or starch dressing, which may be easily re-

moved, should be worn during the after-treatment to prevent the contractures which are apt to develop.

In some cases amputation is better than resection. Amputation is especially indicated in old people, in patients with tuberculosis of the viscera, in extensive involvement of bone, and in those cases in which there is secondary infection with pyogenic bacteria.

According to Poncet, there is an articular rheumatism which is of tuberculous origin. It develops in tuberculous subjects and presents different clinical pictures, occurring in acute, chronic, and anchylosing forms. It is probably due to the toxins of tubercle bacilli or to attenuated forms of the same (Mohr). Lexer once saw an acute form of this arthritis follow an extirpation of tuberculous lymph nodes, a general miliary tuberculosis developing soon afterwards.

(h) TUBERCULOSIS OF TENDON-SHEATHS AND BURSÆ

The clinical course of this form of tuberculosis is as protean as that of tuberculous arthritis. In spite of the many transitions four principal forms may be differentiated:

1. Serous tuberculous hydrops, tuberculous hygroma: Synovial membrane covered with tuberculous granulation tissues. Pure serous exudate.

2. Serofibrinous tuberculous hydrops, rice-body hygroma: Synovial membrane covered with tuberculous granulation tissue, or with villous, fibrinoid growths, little serous exudate, few or many corpora oryzoidea (cf. Joints).

3. Granulating form with cicatrization, fungus: The connective tissue of the sheath is transformed into tuberculous granulation tissue varying from 1 to 2 cm. in thickness. There is little or no exudate; occasionally nodular masses of granulation tissue develop, which may become as large as a pigeon's egg.

4. Granulating form with suppuration, cold abscess: Walls of sheath covered with caseated granulation tissue. Pus and granulation tissue, which may rupture externally, are present in the sheath.

All these different forms may be primary or secondary to some neighboring focus in bone or joint. They may develop in one or many sheaths and may also occur symmetrically. Tuberculous tendo-vaginitis is most frequent in the upper extremities, the large synovial sheaths of the flexor tendons at the level of the wrist joint and of the extensor communis digitorum on the dorsum of the hand being most frequently involved. In the foot the disease attacks most frequently the sheaths of the extensor and peroneal muscles. Primary tuberculous bursitis may develop in any bursa. It is much rarer than secondary tuberculous bursitis.

Clinical Course.—The clinical course is, as a rule, chronic. The first symptoms are radiating pain, limitation of motion, weakness of the part involved, and the development of a long, flat swelling along the tendon. The connection with the tendon is indicated when movements are made. In the forearm and palm an hour-glass swelling is often produced by the constriction of the annular ligament, beneath which the fluid passes readily from one swelling to the other.

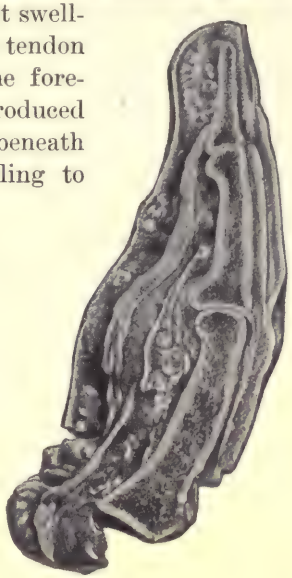

FIG. 186.—TUBERCULOSIS OF THE TENDON-SHEATH OF THE FLEXOR TENDONS OF THE INDEX FINGER (GRANULATING, CICATRIZING FORM). Granulation tissue raises the flexor tendons some distance from the bone.

Diagnosis.—The diagnosis of secondary tuberculous tendo-vaginitis, when there are evidences of tuberculosis of a neighboring bone or joint, is not difficult. It may be difficult, however, to make a diagnosis in the primary forms. The rice-body hygroma may be recognized by the grating of the fluctuating contents of the swelling, and the more rare suppurating form, if fistulæ are present or abscesses are about to rupture, can scarcely be confused with any other lesion. On the other hand, it is difficult to distinguish the pure tuberculous hydrops from that due to other lesions (trauma, chronic irritation, rheumatism, gonorrhea, syphilis). Mistakes may easily be made if there are no other evidences of tuberculosis. [Frequently a microscopic examination of the contents and animal inoculation must be made before a definite diagnosis is possible.] In the granulating, non-suppurating form of tuberculous tendo-vaginitis, a pseudo-fluctuating or firm, resistant swelling develops along the course of the tendon-sheath. The rare lipoma arborescens has much the same clinical appearance. Firm nodules in the walls of the tendon-sheaths may be mistaken for tumors. It is often difficult to differentiate tuberculosis of deep-lying bursæ in which there are masses of granulation tissue from a tumor.

Treatment.—The following fundamental principles should be followed in the treatment of these forms of tuberculosis. In the serous form, with but little thickening of the synovial sheath, the exudate should be removed by aspiration and iodoform-glycerin emulsion should then be injected; the other forms should be operated upon. A long incision should be made corresponding to the long axis of the swelling, the synovial sheath incised, and the fluid contents allowed to escape. The parietal and visceral layers of the synovial sheath, with

the suppurating fibrous tissue and the superficial layers of the diseased tendon should then be removed. After the sutures have been inserted and tied, from 5 to 10 c.c. of iodoform-glycerin emulsion should be injected between the sutures. Active and passive motion and massage should be begun as early as ten days after the operation (providing the tendons are not extensively involved and fibrillated), as a good functional result is obtained earlier in this way.

(i) TUBERCULOSIS OF SEROUS CAVITIES AND DIFFERENT VISCERA

These forms of tuberculosis will be but briefly mentioned and merely from the viewpoint of surgical treatment. Tuberculous pleurisy most frequently demands surgical interference because of the pressure which the exudate exerts upon the lung. The operations usually performed are puncture with aspiration, incision through an intercostal space, or resection of a rib with drainage. Tuberculosis of the pleura is rarely primary, being, as a rule, secondary to tuberculosis of the lungs, vertebræ, or ribs, or developing in the course of a general miliary tuberculosis.

Attempts have been made to cure tuberculous peritonitis, which, in children especially, is secondary to tuberculosis of the mesenteric and retroperitoneal lymph nodes, of the intestines, or of the abdominal viscera, or which results from infection through the blood stream occurring after operative procedures. The favorable results following laparotomy in many cases is due to the passive hyperæmia which is induced, and to the removal of the exudate.

Tuberculosis of the kidney, the urogenital apparatus, the breast, the thyroid gland, and large, solitary cavities in the lung, if favorably situated, may be cured by operative procedures.

GENERAL TREATMENT IN LOCAL TUBERCULOSIS

The general treatment, improvement of nutrition, should never be neglected in the surgical treatment of local tuberculosis.

The general condition of the patient improves most rapidly when good, nutritious, and easily digestible food is supplied. The patient should seek a favorable climate as soon as possible. Those climates are most suitable which permit of an out-of-door life and an abundance of good, fresh air. The patient should not return home and assume family relations again, as other members of the family may be suffering with the same disease, or the quarters may be cramped and the hygiene poor. The children of poor people and poor patients should go to sanitaria or state institutions which are devoted to the treatment of this disease. Mountain and sea air or residence in the South are to

be especially recommended. Sun baths also have a favorable influence upon the general condition.

There are no specific remedies for tuberculosis. Tuberculin, of which so much was expected at one time, has no therapeutic value. Of the many drugs which have been recommended, the creosote preparations are still preferred. [Although tuberculin has of late years fallen into disrepute as a therapeutic agent, attention has again been attracted to it by the work of Wright and his colleagues. Minute doses of tuberculin seem to have a favorable effect upon some cases of tuberculosis, and may be tried to advantage, 0.001 mg. being injected once a week. The work which has been done of late seems to indicate that it is not necessary to take the opsonic index in these cases, the tuberculin being injected once a week and controlled by the condition of the patient.]

ACUTE GENERAL MILIARY TUBERCULOSIS

Tubercle bacilli may invade the blood stream in large enough numbers to produce an eruption of miliary tubercles in a number of the viscera and in the different tissues of the body. This form of tuberculosis, which proves fatal in a short time, is of no surgical significance. The larger viscera, the bones, joints, and the serous cavities are most frequently involved.

Tuberculosis of a large lymph gland adjacent to some large lymphatic vessel, such as the thoracic duct, may extend to the latter. Tuberculous foci then develop in the thoracic duct and countless numbers of bacilli are carried into the blood, and a miliary tuberculosis of the lungs and other viscera then develops. Ponfick was the first to describe tuberculosis of the thoracic duct. The involvement of the radicals of the pulmonary veins (Weigert) and arteries in the tuberculous process is an important etiological factor in this form of the disease. Large groups of bacilli are carried into the blood when caseation of the nodules in the walls of these vessels occurs or thrombi become detached.

Tubercles may develop in the vessel wall proper in two ways: bacilli may be deposited upon the intima (endarteritis tuberculosa, Benda) and in the vasa vasorum, or the tuberculous process may extend to the walls of the larger vessels from an adjacent focus (periangitis tuberculosa). The tuberculous process may extend from foci in bronchial and mesenteric lymph nodes, in the lungs, bones, and joints, even from a focus in the skin (Demme). An acute, general, miliary tuberculosis may follow an operative procedure upon some tuberculous focus, or a subcutaneous injury of tuberculous tissues (König, Wittmer).

Clinically there are differentiated the typhoid, pulmonic, and meningeal types of miliary tuberculosis, depending upon whether the

intestinal symptoms (diarrhœa, intestinal hæmorrhages), the pulmonic symptoms (dyspnœa, cyanosis, cough), or the meningeal symptoms (convulsions, rigidity of the neck, loss of consciousness, delirium) are most prominent. Acute general miliary tuberculosis is accompanied by a continuous fever.

The patient may survive a few hours or many months. Recovery is rare.

LITERATURE.—*Baumgarten.* Ueber Immunisierungsversuche gegen Tuberkulose. Berlin. klin. Wochenschr., 1904, p. 1124.—*v. Behring, Römer* und *Ruppel.* Tuberkulose. Beitr. zur experim. Therapie, 1902, Part 5.—*Bier.* Hyperämie als Heilmittel. Leipzig, 1906.—*Blauel.* Ueber das Reiskörperhygrom der Bursa subdeltoidea. Beitr. z. klin. Chir., Bd. 22, 1898, p. 743.—*v. Bruns.* Ueber die Ausgänge der tuberkul. Koxitis bei konservat. Behandlung. Chir.-Kongr. Verhandl., 1894, I, p. 1.—*Cornet.* Die Tuberkulose Miliartuberkulose, Skrophulose. Wien, 1899, in Nothnagel's spez. Pathol. und Therap., mit vollständ.—*Cornet* und *A. Meyer.* Tuberkulose. In Kolle-Wassermann's Handb. der pathog. Mikroorganismen, Bd. 2, 1903, p. 78.—*v. Dungern.* Beitr. z. Tuberkulosefrage auf Grund experim. Untersuch. an anthropoiden Affen. Münch. med. Wochenschr., 1906, p. 4.—*Friedmann.* Experim. Beitr. zur Frage kongenitaler Tuberkelbazillenübertragung u. kongenitaler Tuberkulose. Virchow's Archiv, Bd. 181, 1905, p. 150. —*Friedrich.* Experim. Beiträge zur Kenntnis der chir. Tuberkulose. Deutsche Zeitschr. f. Chir., Bd. 53, 1899, p. 512.—*Garré.* Zur Aetiologie der kalten Abszesse. Deutsche med. Wochenschr., 1886;—Die primäre tub. Sehnenscheidenentzündung. Beitr. z. klin. Chir., Bd. 7, 1891, p. 293;—Ueber die Indikationen zur konservativen u. operativen Behandlung der Gelenktuberkulose. Deutsche med. Wochenschrift, 1905, p. 1878.—*Gebele* und *Ebermayer.* Ueber Behandlung bei Gelenktuberkulose. Münch. med. Wochenschr., 1906, p. 601.—*Grober.* Die Tonsillen als Eintrittspforten f. Krankheitserreger, bes. f. d. Tuberkelbazillen. Klin. Jahrb., Bd. 14, 1905, Part 6.—*Henle.* Die Behandlung der tub. Gelenkerkrankungen und der kalten Abszesse. Beitr. z. klin. Chir., Bd. 20, 1898, p. 363.—*Otto Hildebrand.* Tuberkulose und Skrophulose. Deutsche Chir., 1902.—*Honsell.* Ueber Trauma und Gelenktuberkulose. Beitr. z. klin. Chir., Bd. 28, 1900, p. 659.—*Jacob* und *Pannwitz.* Entstehung und Bekämpfung der Lungentub. Leipzig, 1901.—*Jordan.* Ueber Tuberkulose der Lymphgefässe der Extremitäten. Beitr. z. klin. Chir., Bd. 19, 1897, p. 212.—*R. Koch.* Ueber neue Tuberkulinpräparate. Deutsche med. Wochenschr., 1897, p. 209.—*König.* Die Tuberkulose der Knochen und Gelenke. Berlin, 1884;—Die spez. Tuberkulose der Knochen und Gelenke: I. Das Kniegelenk. Berlin, 1896; II. Das Hüftgelenk. Berlin, 1902;—Die Tuberkulose d. Thoraxwand. Arch. f. klin. Chir., Bd. 79, 1906, p. 1.—*Krause.* Die Tuberkulose der Knochen und Gelenke. Deutsche Chir., 1899. —*Küttner.* Die Osteomyelitis tub. des Schaftes langer Röhrenknochen. Beitr. z. klin. Chir., Bd. 24, 1899, p. 449.—*Lanz* und *de Quervain.* Ueber hämatogene Muskeltuberkulose. Arch. f. klin. Chir., Bd. 46, 1893, p. 97.—*Lexer.* Weit. Untersuch. über Knochenarterien und ihre Bedeutung f. krankhafte Vorgänge. Arch. f. klin. Chir., Bd. 73, 1904, p. 481.—*Lorenz.* Die Muskelerkrankungen. In Nothnagel's spez. Pathol. und Therap. Wien, 1898.—*Masur* und *Kockel.* Wirkung toter Tuberkelbazillen. Ziegler's Beitr. z. path. Anat., Bd. 16, 1894, p. 256.—*Mohr.* Der Gelenkrheumatismus tuberk. Ursprungs. Berliner Klinik, 1905, Part 197.—*Friedrich Müller.* Ueber die Bedeutung der Selbstverdauung bei einigen krankhaften Zuständen. 20. Kongress der inn. Mediz., 1902.—*Orth.* Perlsucht und menschl. Tuberkulose? Berl. klin. Wochenschr., 1903, p. 657.—*Orth* und *Esser.* Was ist Perlsucht? Berl. klin. Wochenschr., 1902, p. 793.—*Ostertag.* Koch's Mitteilungen über die Beziehungen der Menschen- zur

Haustiertuberkulose. Zeitschr. f. Fleisch- und Milch-hygiene. Berlin, 1901, Part 12. —*Roger* et *Garnier*. Passage du bacille de Koch dans le lait d'une femme tub. Compt. rend. de la Société de biolog., 1900, p. 175.—*Schmorl* und *Geipel*. Ueber die Tuberkulose der menschl. Plazenta. Münch. med. Wochenschr., 1904, p. 1676.—*Sternberg*. Experim. Untersuchung. üb. die Wirkung toter Tuberkelbazillen. Centralbl. f. allg. Pathol., Bd. 13, 1902, p. 753.—*v. Székely*. Neuere Arbeiten über die Frage der Identität der menschl. und der Rindertuberk. Centralbl. f. Bakt., Bd. 34, 1903, Ref., p. 161.—*Tavel*. Beiträge zur Aetiologie der Eiterung bei Tuberkulose. Festschrift für Kocher, Wiesbaden, 1891.—*v. Volkmann*. Lupus und Tuberkulose. Berl. klin. Wochenschr., 1875, p. 413.—*Wassermann*. Tuberkulose im frühesten Kindesalter. Zeitschr. f. Hygiene, Bd. 17, 1894, p. 343.—*A. Wassermann* und *Bruck*. Exper. Studien über die Wirkung von Tuberkelbazillenpräparaten au den tuberkulös erkrankten Organismus. Deutsche med. Wochenschr., 1906, p. 449.—*A. Weber*. Die Tuberkulose des Menschen und der Tiere. In Kolle-Wassermann's Handb. d. pathog. Mikroorg., 1906. Ergänzungsband 1, p. 107.—*Wittmer*. Ueber akute Miliartuberkulose nach Operation tuberkulöser Halsdrüsen. Beitr. z. klin. Chir., Bd. 33, 1902, p. 788.—Handbuch der praktischen Chirurgie von *v. Bergmann, v. Bruns*, und *v. Mikulicz*. Abschnitte von *Henle, Hoffa, Jordan, Lexer, Reichel*.—Internation. Chir.-Kongress z. Brüssel, 1905. Traitement de la tuberculose articulaire.

CHAPTER XI

LEPROSY (ELEPHANTIASIS GRÆCORUM)

Geographical Distribution and Course of Extension.—[Ricketts gives the following sketch of the course of extension of leprosy: "Leprosy existed in Egypt in prehistoric times and extended to other lands only when intercourse was established between the two countries. It reached Greece at about 345 B.C., Italy in the first century before Christ, and from the latter country extended to Germany, France, and Spain. Crusaders returning from the Orient also brought back the disease in later times and eventually all Europe was infected. Leprosy is known to have existed in Great Britain in the tenth century, and from that country it was carried to Ireland and Greenland. From Germany it extended to the Scandinavian countries, and from the latter to Finland and Russia. It also reached Russia from the south and east, and in the south it was at one time called the Crimean disease. The West Indies and South America probably were infected from Spain, and through these channels the disease was carried to the Southern States of America. The leprosy of the Western States seems to have been imported by Norwegian immigrants chiefly. In 1902 the United States Leprosy Commission found 278 cases in this country; 186 of these individuals probably contracted the disease in this country, 120 were born in foreign countries, and 145 were native born. The disease also extended around the globe in the opposite direction, reaching China,

Japan, and the East Indian islands from India. The Sandwich Islands became infected in the nineteenth century.

"The contagiousness of the disease appears to have been recognized at a very early period. In 636 A.D. leprosy houses were instituted in Italy and other countries, and the practice of segregating lepers soon became general. The hospitals were called Lazarus houses in Middle Europe, and St. George houses in Scandinavian countries. Pepin and Charles the Great declared marriage between lepers illegal. The rapid disappearance of leprosy in Middle Europe during the sixteenth century is ascribed largely to the segregation of patients."]

Bacillus of Leprosy.—The bacillus of leprosy was first demonstrated by Hansen, later by A. Neisser. In form, size, and staining reaction, both in dry preparations and sections, it resembles closely the tubercle bacillus.

Bacilli of leprosy occur in groups, while the micro-organism of tuberculosis occurs singly or in small clusters and outside of the cells. Leprosy bacilli take the stain more readily than tubercle bacilli, and for this reason may be stained with cold solutions. Baumgarten recommends the use of a dilute, cold solution of fuchsin (five drops of a saturated alcoholic solution in a watch crystal full of water). The preparations are stained from six to seven minutes in this solution, are then detained for fifteen seconds in a ten per cent solution of nitric acid in alcohol, and are counterstained, after the acid-alcohol is removed with water, in a dilute solution of methylene blue. In this method the bacilli of leprosy appear red, while the tubercle bacilli are unstained.

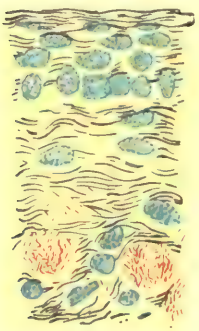

FIG. 187.—BACILLI OF LEPROSY IN THE SKIN. (After A. von Bergmann.)

They are found in largest numbers in the cellular, inflammatory growths following infection, and in the mucous membranes. They may be demonstrated in the diseased peripheral nerves, in the lymph nodes, and viscera (liver, spleen, lung, and testicle). Bacilli have also been found in the spinal ganglia and cord, upon the surface of the skin and mucous membranes (Babes). They may be found in the blood during life, if the blood is examined during the febrile period which frequently accompanies the development of new nodules. The attempts which have been made to cultivate the bacilli upon artificial media and to produce the disease by inoculating animals have not been successful. Animals are not susceptible to leprosy, and therefore it has been difficult to identify the cultures which some have claimed to obtain. In spite of these facts a direct or indirect transference of the

disease from a diseased to a healthy subject can no longer be doubted, for the bacilli may be demonstrated in inflammatory nodules and infiltrations. The danger of infection does not, however, seem to be great.

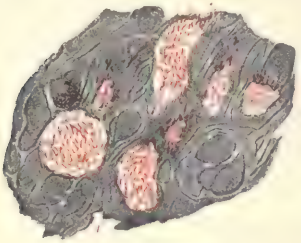

FIG. 188.—LEPROSY BACILLI IN THE MUCOUS MEMBRANE. (After A. von Bergmann.)

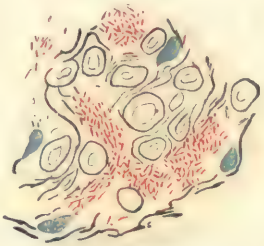

FIG. 189.—LEPROSY BACILLI IN THE NERVES. (After A. von Bergmann.)

The disease is not inherited, but is transmitted from one member of the family to another through exposure.

Modes of Infection.—The bacilli gain entrance in some unknown way, apparently through the skin or mucous membrane, and are then carried to other parts through the lymphatic vessels or blood vessels. Wherever they are deposited they produce a chronic inflammation, resulting in the formation of a vascular granulation tissue. Nodular inflammatory growths develop about the hair follicles (Virchow), later appearing as large, flat, infiltrated areas and nodules. These inflammatory changes occurring in the nerves produce spindle-shaped thickenings, the connective-tissue fibers of the nerves become stretched, and the nerve fibers are destroyed. All these pathological changes are grouped under the term LEPROMA. The bacilli lie in large groups within and without the cells, also in multinuclear giant-cells. They may be easily demonstrated in blood discharged from nodules and from the skin after it is pricked. Large vacuolated cells, called *globi* or lepra cells, filled to bursting with degenerating bacilli (Virchow, Neisser, Hansen), are supposed to be characteristic of the disease. These masses are regarded by some as bacillary thrombi in lymphatic and connective-tissue spaces. The nodules may ulcerate; occasionally they may become absorbed. Frequently they remain unchanged when the height of their development is reached.

Period of Incubation.—Years may intervene after the infection has occurred before symptoms of the disease develop. This is demonstrated by the fact that the people who have lived in leprous regions may develop the disease some time after they have returned home where the disease does not exist. In these cases the symptoms develop on an average from three to five years afterwards, although isolated cases

have been observed in which the symptoms developed after a much longer period (twenty-seven to thirty-two years).

Symptoms and Clinical Course.—The most important, but not the most constant, symptoms in the beginning of the disease are the sensation of intense cold in the hands and feet, hyperæsthesia, especially of the lower extremities, and erythematous spots which may appear upon the surface of the entire body and disappear after a few days without leaving any trace to soon reappear. According to A. von Bergmann, in some of the cases flat, slightly raised, infiltrated areas, varying from a dull red to a brownish red color and several centimeters in diameter may be demonstrated in the skin. These may be the only, though positive, signs of both forms of leprosy, and may last for a number of years. According to Sticker, Kolle, and others, leprosy begins in a number of cases in the nasal mucous membrane.

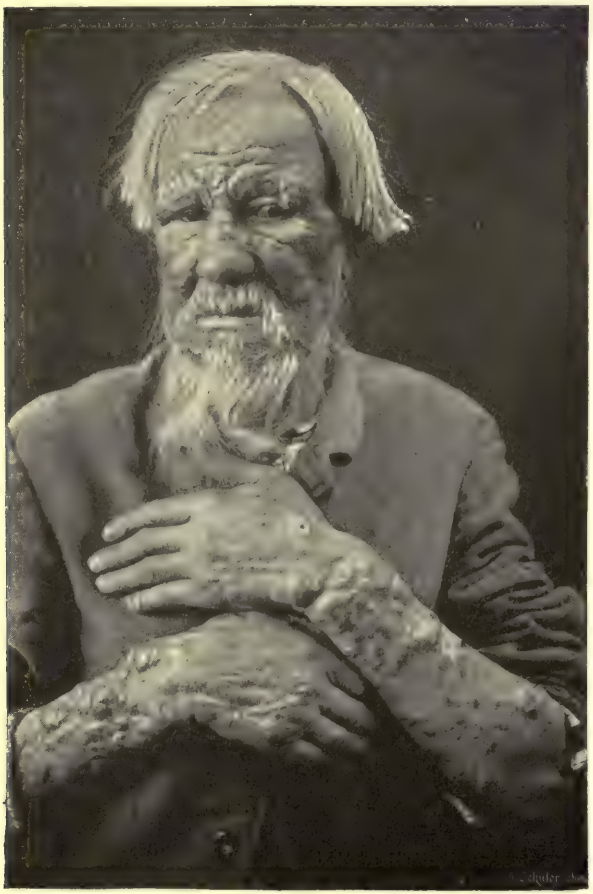

FIG. 190.—LEPRA TUBEROSA. (After A. von Bergmann.)

The disease occurs in two forms, depending upon whether the pathological changes are most marked in the skin or nerves. These two forms may be combined.

Leprosy of the skin (lepra tuberosa) begins with the formation of small nodes, some of which develop from the dull red or brownish red spots above described. These give to the skin an uneven, tuberculated appearance. General symptoms and fever, caused by the diffusion of

bacilli and their deposition in the capillaries of the skin, often accompany these local changes. While the nodes upon the face and the dorsum of the hands, where they first develop most frequently, fuse to form large hyperæmic spots and infiltrated areas and masses, which are covered with pigmented skin or develop by gradual growth into round, soft, red and bluish nodes or masses the size of a walnut with broad bases, new nodes develop, accompanied by a febrile reaction, upon other parts of the body, especially upon the face and the extensor surfaces of the extremities. The most marked changes are always found in the skin of the face. Nodes and inflammatory masses develop upon the forehead, the nose, the cheeks, and the ears, while the beard, the eyelashes, and eyebrows fall out. The masses which develop in the skin are traversed by the natural folds of the latter. When these changes are advanced, the patient has the facial expression of an animal, and hence the expressions *satyriasis* and *leontiasis*, to describe this condition, used by the ancients. The tumorlike nodes may develop upon the nose, the eyebrows, and the lips, so that often in addition to the deformity above described the mouth and the palpebral fissures may become narrowed. Inflammatory masses which have remained unaltered for a long time may, after a febrile reaction, swell and soften and then disappear or ulcerate.

Leprosy of the Mucous Membranes.—The mucous membranes are not spared. At the same time that the eruption occurs in the skin, the conjunctiva (with extension to the cornea and iris) may become involved, and nodes and inflammatory masses may develop in the mucous membrane of the nose, buccal cavity, and pharynx (especially upon the soft palate), and in the larynx.

Involvement of the latter produces not only the raucous voice of leprosy (Vox rauca leprosa), but frequently also laryngeal stenosis.

Chronic ulcers of the skin and mucous membranes follow the breaking down of these nodes and masses. Ulcers of the leg are frequent in leprosy, and the demonstration of bacilli in their secretion is proof positive that they have followed the ulceration of preëxisting nodes. The lymph nodes adjacent to the diseased area may become swollen and reach quite a considerable size. A chronic periostitis may give to the bones of the part involved a spindle-shaped enlargement (de la Camp).

The clinical course of the disease varies. In one patient isolated and unaltered nodes may be the only indication of the disease; in another new inflammatory masses and ulcers develop one after another; and in still another case there may be a transition to the nervous form of leprosy. The patient becomes a chronic invalid and death occurs in from five to nine years, the result usually of leprosy or of amyloid

degeneration of the important viscera. It is frequently due to extensive involvement (partly leprous, partly tuberculous) of the lungs.

In the *nervous form of leprosy* (lepra nervorum, s. maculo-anæsthetica), flat, brownish-red, infiltrated areas develop in the skin. These may develop independently without symptoms or from preëxisting areas. The somewhat elevated and pigmented border of such an area extends into the surrounding tissues, while the center of the area becomes depressed and white, and the hair falls out. The skin becomes atrophic, and the first evidence of disturbance of nerve function (*anæsthesia*) is found in these white areas. These areas which, formerly depending upon whether they were pigmented or not, were spoken of as white and black leprosy (morphœa alba and nigra), may be very extensive and often extend symmetrically resembling in outline a map. According to A. von Bergmann, in the nervous form of the disease bacilli are not found in the flat, infiltrated areas. According to Danielssen and Babes, only a few bacilli are present, and it is difficult to demonstrate them.

FIG. 191.—LEPRA MACULO-ANÆSTHETICA.
(After A. von Bergmann.)

The evidences of nerve involvement are most frequently seen first along the course of the ulnar, median, peroneal, and facial nerves. The sciatic and femoral nerves may become diseased. Sensory disturbances are the most prominent. Bacilli and mild inflammatory changes have been found even in the spinal and Gasserian ganglia and in the spinal cord. The nerves, when involved, become thickened, and may be palpated as swollen cords with spindle-shaped thickenings. The

areas supplied by the diseased nerves become anæsthetic and subject to a number of trophic disturbances, such as: (1) Atrophy of groups of muscles with motor paralysis. Atrophy of the muscles of the thenar and hypothenar eminences, of those upon the ulnar side of the forearm, of the face and neck, and in the interossei spaces occurs earliest and is most common. (2) Contractures of the fingers (fourth and fifth most frequently) and toes; also of the forearm and leg. (3) Ulcers due to pressure, which are round and develop upon the sole of the foot beneath the heel and the heads of the metatarsal bones. (4) Mutilation of the fingers and toes (lepra mutilans) following necrosis of the phalanges; mutilation by ulcers, accompanied by a progressive necrosis of the connective tissues, so that separate digits or the entire hand and foot may slough off at one of the joints. In these cases small, independent foci of leprous granulation tissue develop in the phalanges (Sawtschenko). (5) Vesicular eruptions (pemphigus leprosus) develop most frequently upon the extremities. These eruptions, which are followed by chronic ulcers, recur at irregular intervals.

Diagnosis.—The diagnosis of leprosy occurring in regions where it is not endemic is very difficult in the early stages of the disease. Lepra mutilans resembles closely syringo-myelia, which is accompanied by mutilation of the fingers, sensory disturbances, and atrophy of muscles. Frequently it is scarcely possible to differentiate between the two, for it is often difficult to demonstrate the thickening of the nerves, which is the only definite characteristic of lepra nervorum. In all doubtful cases examination of the nasal secretion for the bacillus of leprosy should be made.

Treatment.—Patients suffering with leprosy should be isolated. They should receive good nursing and good food. Cleanliness should be exercised in caring for the patient and his surroundings. Not infrequently surgical interference is required. The troublesome and disfiguring tumors developing upon the face may be removed. Fingers and toes, the seat of a progressive and persistent necrosis, should be amputated or disarticulated. If stenosis of the larynx develops, it may be necessary to perform a tracheotomy. Ulcers developing upon the legs and soles of the feet should be dressed aseptically and protected from further injury. Danielssen recommends sodium salicylate internally; von Bergmann, Gurjun balsam (5 drops daily), also an external application of the same (2.0–3.0 daily) with lanolin used as a salve.

LITERATURE.—*Babes.* Die Lepra. Spez. Pathologie und Therapie von Nothnagel, Bd. 24, Wien, 1901;—Lepra. In Kolle-Wassermann's Handbuch der pathog. Mikroorganismen. Ergänzungsband, 1906, p. 155.—*A. v. Bergmann.* Die Lepra. Deutsche Chir., 1897.—*de la Camp.* Periostitis bei Lepra. Fortschritte auf dem Gebiete der Röntgenstrahlen, Bd. 4.—*Hansen.* Lepra. In Kolle-Wassermann's Hand-

buch der pathog. Mikroorganismen, Bd. 2, 1903, p. 178.—*Uhlenhut* und *Westphal.* Hist. u. bakteriol. Untersuchungen über einen Fall von Lepra tuberosa-anaesthetica. Centralbl. f. Bakteriol., Bd. 21, 1901, p. 233.—*Virchow.* Die krankhaften Geschwülste, Bd. 2, p. 494.

CHAPTER XII

SYPHILIS

Spirochæta Pallida.—Syphilis (lues) belongs to the chronic infectious diseases. The micro-organism discovered by Schaudinn and E. Hoffmann in 1905, which is known as the spirochæta pallida, is apparently the cause of the disease. The organism measures from 4 to 14 μ in length, the average length being about the diameter of the erythrocyte of man. Its width varies from immeasurable thinness to 0.5 μ. It possesses from three to twelve, sometimes more, curves, which are sharp and regular and resemble the curves of a corkscrew. The poles are sharpened. The organism is motile, the motions consisting of rotations on the long axis, forward and backward movements, and the bending of the entire body. Flagella have not been seen (Flexner). It stains with the azur of Giemsa and with aniline dyes, only after being acted upon for some time, however.

FIG. 192.—SPIROCHÆTA PALLIDA. *a*, red; *b*, white blood corpuscles.

These organisms are found in the ulcerated, more rarely in the non-ulcerated chancre and papules, in the roseola, in the circulating blood, and in the lymph nodes. In congenital syphilis they are found in almost all the viscera. Their presence in the ulcers of late syphilis has not been satisfactorily demonstrated. The spirochæta has been found in syphilis produced experimentally in apes (Metschnikoff and others). Attempts at cultivating them have not been successful.

Special text books have been devoted to syphilis, the clinical course and symptoms of which differ very much in different individuals. Every physician should be thoroughly conversant with the pathology,

clinical course, complications, and sequelæ of the disease. The greater part of the treatment belongs to internal medicine.

The *initial lesion* (initial sclerosis, Hunterian induration, hard chancre) develops at the point where the syphilitic virus, which is discharged from an ulcerated chancre or a moist papule developing in the early stages of the disease, gains access to a small wound, or an erosion of the skin or mucous membrane. The virus may be transferred directly by contact of the syphilitic lesion with the wound or excoriation, or indirectly by the finger or some object (e. g., drinking and eating utensils, cigar holder, needle used for tattooing).

The extra-genital chancre may develop on any part of the body. It occurs most frequently upon the face (lip, nose, canthus of the eye, tonsil, tongue), the fingers, and breast. The diagnosis is much more difficult than when the chancre occurs upon the genitalia or structures adjacent to them.

After an incubation period of from two to four weeks a papule or an induration, depending upon whether the changes are superficial or deep, develops about the infection atrium, which in the meantime may have healed or have become transformed into a deep ulcer. If there is a mixed infection, a soft chancre may develop within two days, and then the induration, so characteristic of the hard chancre, develops in the soft chancre after the usual incubation period.

Appearance of the Chancre.—The initial lesion appears as a round or oval nodule. It is red, sharply delimited, hard, painless, and may become as large as a quarter. Often the initial lesion is so small, especially in women, that it may be overlooked. An urethral chancre frequently is overlooked, being accompanied merely by a slight urethral discharge, and the development of the syphilitic eruption may be the first indication of a preceding infection. If the epidermis covering the chancre is cast off, the ulcerated surface becomes covered with a firmly attached crust. When the crust is removed bleeding occurs. If the lesion is situated upon mucous or skin surfaces, which are moist, the surface of the lesion does not become covered with a crust, but secretes continuously (moist papule, ulcerated sclerosis of the genitals, anus, and buccal mucous membrane). The disintegration of the infiltration, which is often hastened by the use of

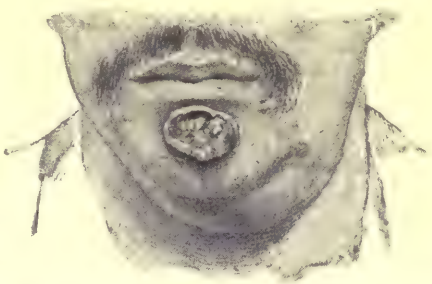

Fig. 193. — An Ulcerated Chancre Ten Days Old. The lesion, which followed a razor cut, is accompanied by a marked enlargement of the regional lymph nodes.

caustics, uncleanliness, etc., leads to the formation of an ulcer. The ulcer has a hard, parchmentlike base, and slightly raised, indurated, wall-like borders, which are not undermined. In rare cases the initial sclerosis is complicated by gangrene; a deep phagedenic ulcer covered by a dark crust then develops.

Disappearance.—The primary lesion disappears very slowly after weeks or months. The amount of induration gradually diminishes and the cells undergo fatty degeneration and become absorbed.

Histology.—Histologically there is found in the primary lesion a collection of round and epithelioid cells with an occasional giant-cell. The pathological changes occur first about the vessels; finally they involve the entire thickness of the skin or mucous membrane. The lumina of the blood vessels may become obliterated by the proliferation of the tissues composing them, especially by a proliferation of the intima (endovasculitis obliterans syphilitica). Frequently these vascular changes are the beginning of regressive changes in the primary lesion.

Diagnosis.—The induration of the primary lesion is its most important characteristic, and is often the deciding factor in making a diagnosis. An ulcerated chancre may resemble clinically a carcinoma of the skin. In the chancre, however, the induration and ulceration develop much sooner than in a carcinoma, and the involvement of lymph nodes is much more rapid and extensive in relation to the size of the lesion than is the case in carcinoma of the skin. Enlargement of lymph nodes does not occur with an ulcerated gumma, unless there is a secondary infection. This enables one to differentiate between an ulcerated gumma and a chancre of a mucous membrane.

Treatment.—Early excision of the primary lesion is unreliable, although at one time it was hoped that the absorption of the organism and the development of a general infection might be prevented by this procedure. Excision is not employed at the present time except in rare instances. If excision is performed it should be done before there is any enlargement of the adjacent lymph nodes, and only in the first week after the development of the lesion. It should be kept in mind that the fresh wound following excision may become infected with the secretion of the chancre.

All that is necessary in the treatment of the chancre is to keep the lesion as clean as possible and to avoid irritation and injury. Aristol, dermatol, bismuth subnitrate, or some other dusting powder may be used and a gauze dressing applied. [Caustics should never be employed in the treatment of lesions upon the penis. The induration which follows the use of caustics may render the diagnosis of doubtful lesions impossible. The healing of a primary lesion is not favored by the use of any of these agents. Cleanliness of the part with the use

of some mild ointment or powder as above mentioned is indicated. Mercury preparations should be avoided in the treatment of any suspected lesion, as the appearance of the initial lesion may be so changed and the subsequent clinical course of syphilis so modified that it may be impossible to make a correct diagnosis.] The general treatment should be begun with the first symptoms of the secondary stage.

Involvement of Adjacent Lymph Nodes.—The lymph nodes adjacent to the chancre enlarge slowly from four to five weeks after the infection, from one to two weeks after the development of the primary lesion, and become as large as a cherry or nut. They are painless, hard, and not adherent to the skin or to neighboring nodes, and form adhesions with adjacent structures only when secondary infections occur. Involution of these lymph nodes is extremely slow, and they remain enlarged for a long time after the primary lesion has completely healed. Enlargement of nodes adjacent to the parts upon which the primary lesion is most frequently located may be of value in making the diagnosis of previous infection. The inguinal nodes are the most frequently involved, as the genital is much more frequent than the extra-genital chancre. The enlarged nodes (indolent bubo) lie arranged in a row (syphilitic rosary) in both inguinal regions following an initial lesion on the penis.

Eruptive Stage.—Syphilis is a local disease until the lymph nodes adjacent to the chancre become enlarged; then, after a second incubation period of some weeks, the general infection of the body is indicated by the development of different lesions of the skin and mucous membranes (eruptive stage). General symptoms, such as pain in the head and extremities, weakness, and mild fever, precede and accompany the eruptive stage. At the same time all the palpable lymph nodes become enlarged (scleradenitis). They may be felt as hard nodules as large as a bean and persist for years.

In the eruptive stages a macular, papular, pustular, or scaly eruption develops upon the skin and mucous membranes. These different eruptions, which may be combined, are of great diagnostic significance and recur frequently (so-called secondary syphilis).

In congenital syphilis, in which, according to Lesser, the symptoms of the different stages develop in more rapid succession than in the acquired forms, the different forms of eruption above mentioned also occur, and as a rule within the first two months of life.

The late or tertiary symptoms of the disease, both in the acquired and congenital forms, have no definite time limits. They may follow immediately the lesions of the secondary stage, or develop while the secondary lesions are at their height (galloping syphilis). Often they develop after an interval of years, during which time there may have

been no symptoms of the disease (latent period). The tissue changes characteristic of the third stage of syphilis are much more destructive than those of the secondary stage, but the resulting lesions can no longer transmit the disease, as their secretion is no longer infectious. As the late lesions are the ones most frequently treated surgically, it is comforting to know that there is no danger of specific infection.[1]

The Gumma.—The gumma (gummatous tumor, syphiloma, nodular syphilide) is characteristic of late or tertiary syphilis. It is a granulation tumor (Virchow) and is peculiar in that it may develop as a single or multiple lesion in any tissue and in any part of the body. A gumma develops slowly to attain the size of a walnut or fist, and the tissues or the parenchyma of the organ in which the gumma develops are infiltrated or replaced by granulation tissue rich in cells and blood vessels. If regressive changes occur in the nodule, the tissue which has been infiltrated degenerates or is transformed into a mass of scar tissue.

Macroscopic Appearance of a Gumma.—Upon section, a recent gumma is of a grayish or grayish red color, translucent, and sometimes of a gelatinous consistency (soft forms, poor in cells). The great tendency to undergo regressive changes, peculiar to gummata, is due for the most part to the involvement of the walls of the blood vessels by the syphilitic process (vasculitis and perivasculitis syphilitica). These vascular changes frequently precede the degenerative changes in the gumma. As the regressive changes progress, the center of the gumma becomes transformed into a yellowish, opaque focus or appears to be divided into a number of necrotic areas, which may fuse, so that finally the necrosis extends and involves the entire lesion. In the meantime the surrounding connective tissue has proliferated to form a connective-tissue capsule, from which processes extend into the degenerating mass.

Regressive Changes.—The fate of the gumma differs, depending upon the character of the necrosis. Deep gummata frequently are absorbed, disappearing completely, or the necrotic tissue becomes encapsulated. The nodule may become transformed by caseation and coagulation-necrosis into a dry, firm, homogeneous mass, or by fatty degeneration of the cells into a friable one. If the necrotic tissue becomes liquefied, abscesses containing a fluid resembling pus and caseous particles form. If the skin or mucous membrane covering a gumma becomes necrotic, ulcers develop.

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[1] The investigations of A. Neisser have shown that there is still a possibility of infection from the late lesions of syphilis, for he has been able to produce typical primary lesions in apes by inoculating them with tissues taken from the late lesions. The non-ulcerated gummatous lesions were the only ones, however, which gave positive results, and this explains how the false sense of security based upon practical experience that the gummatous lesions in general are not infectious has arisen.

Gummatous Ulcer.—The ulcer which follows regressive changes in a gumma has some very important characteristics. It is round, its edges are sharply cut and but little undermined, and its floor is covered with grayish yellow necrotic tissue, which is sometimes dry and at other times pours out a purulent secretion. The ulceration extends deeply into the firm tissue composing the nodule. When the entire lesion is destroyed by chronic suppuration, and there is no extension of the

FIG. 194.—ULCERATED GUMMA WITH SCALLOPED BORDERS DUE TO UNEQUAL CICATRIZATION.

process from the periphery of the ulcer, healthy granulating tissue develops and the ulcer may heal. If healing occurs upon one side of the ulcer and degenerative changes on the other, reniform, sickle-shaped ulcers develop. If many ulcers are grouped in one region they produce by their confluence lesions with rounded or serpiginous outlines.

Histology of a Gumma.—Microscopically a gumma consists of granulation tissue in which are found epithelioid cells and an occasional giant-cell. The blood vessels, usually diseased, are more numerous than in the tubercle. The walls of the vessels in the gumma are usually thickened and infiltrated with round cells (vasculitis and perivasculitis syphilitica).

According to Kaufmann, three zones may be differentiated when the center of a gumma becomes necrotic: (1) In the center a mass which is caseated or has undergone fatty degeneration; (2) an intermediate zone composed of fibrous connective tissue with spindle cells, isolated epithelioid cells, and giant-cells; (3) an outer zone of proliferating connective tissue, rich in cells and blood vessels.

The alterations in the blood vessels differentiate the gumma from the tubercle and the small round-cell sarcoma. In making a differential diagnosis, the peripheral parts of a gumma, in which most of the vessels lie, should be carefully examined.

Diagnosis.—In considering the general diagnosis of syphilis it should be mentioned that pregnancy is often interrupted by syphilitic disease of the placenta or fœtus. Miscarriages are frequent in syphilitic patients and the fœtus is dead, macerated, or non-viable when expelled. In making a diagnosis of doubtful lesions an accurate history as to previous miscarriages should always be elicited.

The following lesions which occur in congenital syphilis are of great diagnostic importance: (1) Interstitial or parenchymatous keratitis, which develops at puberty or earlier. At first this is limited to one eye, but the other eye will almost certainly become involved later. Ulcers do not develop upon the cornea, but opacities develop as the result of the inflammation. (2) Deformities of the teeth, first described by Hutchinson, occurring most frequently in the central upper incisors, but the upper lateral and the lower incisors may also be involved. The size of the teeth diminishes from root to crown, and they are often separated from one another by wide spaces. A notch occupies the center of the margin of the tooth, or they may be "peg-shaped," the tooth narrowing from the root to the crown and ending in a free border. These changes are found in the teeth of the second dentition, which may appear early, be discolored, and soon crumble away. Similar deformities of the teeth occur in other diseases, associated with severe nutritional disturbances. If deafness develops as the result of involvement of the internal ear and the corneal opacities and deformities of the teeth are present, one speaks of Hutchinson's triad, which, however, is not infallible in making a diagnosis of syphilis. Fine linear scars radiating from about the mouth, which follow the healing of rhagades, are valuable diagnostic signs.

(a) SYPHILIS OF THE SKIN

With the exception of the primary lesions above described, the tertiary lesions of the skin are of the most interest to the surgeon. The secondary lesions are most frequently seen and treated by the dermatologist. Clinically the symptoms of a gumma vary, depending upon whether it is cutaneous or subcutaneous.

Cutaneous gummata are seen frequently in the early stages of tertiary syphilis. They appear as firm, reddish brown, slightly elevated nodules about the size of a split pea, and are frequently arranged in groups. The nodules occupying the center of the group may be ab-

sorbed, or ulcerate and heal, while adjacent healthy tissue about the periphery of the group is invaded by new nodules. If the nodules coalesce, a diffuse infiltration of the skin, appearing as a red, hyperæmic area with a round or serpiginous border, develops (papulo-serpiginous syphilide, Fig. 195). The ulcerated center of such a lesion may heal, while the periphery extends.

The nodules and ulcers occurring in this form of syphilis may be very similar to those found in lupus, and mistakes in diagnosis are not infrequent, especially when the lesions occur upon the face. Lupus develops much more slowly than syphilis, and the nodules develop in the scars resulting from the healing of old ulcers. A tuberculosis lesion does not extend so rapidly from the periphery and does not have the serpiginous outline. The apple-jellylike granulations so characteristic of lupus are not found in the syphilitic lesions. The deep suppurating syphilitic ulcer with its steep, sharply defined borders differs from the flat, somewhat elevated ulcer of lupus with its apple-jellylike granulations.

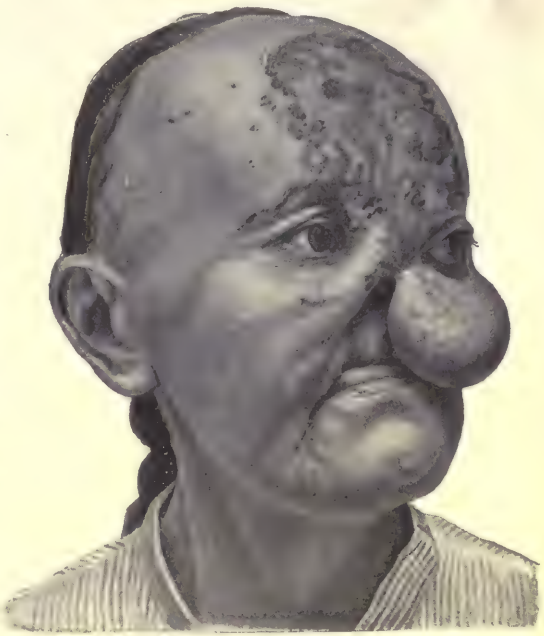


FIG. 195.—PAPULO-SERPIGINOUS SYPHILIDE OF THE SKIN OF THE FOREHEAD. Complete destruction of the nose and upper lip. Chronic œdema of the left lower eyelid.

The *subcutaneous gumma* appears as a round or oval nodule or indefinite thickening of the subcutaneous tissue. It develops slowly without giving rise to any distinct symptoms, to become as large as a walnut or larger. As the gumma approaches the surface the skin covering it becomes red or reddish brown. The subcutaneous nodules are but rarely disseminated over the surface of the body. The skin of the face (nose, forehead, lips) and the thigh are most frequently involved.

Sometimes subcutaneous gummata do not ulcerate, but become absorbed. Frequently, however, ulceration occurs and the resulting lesion is deep, its edges are sharply cut, and its base consists of granulation

tissue, although it is sometimes covered by a characteristic slough. These ulcers, in case they do not extend deeply and destroy more tissue, leave when they heal a white, shining, round scar. Extensive destruction occurs most frequently when these lesions develop upon the face, where not only the lips and eyelids, but also the entire nose with its cartilaginous and bony framework may be destroyed. The scalp and skin of the forehead may be transformed into a large, shining, flat, firmly attached scar, throughout which appear swollen areas of skin (Fig. 196). In severe cases only the unprotected bulbs of the eyes and the openings which indicate the position of the nose and mouth remain within the radially arranged masses of scar tissue. In rare cases a carcinoma develops upon a syphilitic ulcer; then the syphilitic ulcer assumes the characteristics of a carcinomatous ulcer.

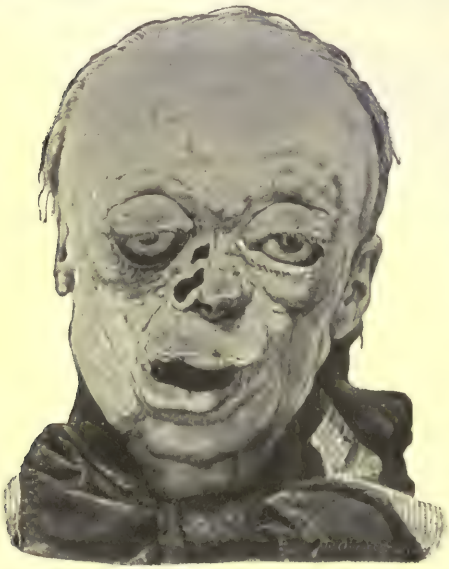


FIG. 196.—A THIRTY YEAR OLD WOMAN INFECTED WITH SYPHILIS. Extensive destruction of the skin and bones of the face. Defects of lids, cheeks and lips repaired by non-pedunculated cutis flaps. Saddle-nose with perforation, anchylosis of the temporo-mandibular joint.

Differential Diagnosis. — The diagnosis of subcutaneous gummata and the ulcers developing from them is not difficult, especially if there are other symptoms of syphilis. As a rule, the lesions are so characteristic that there is no difficulty in making the diagnosis, even when no other lesions are present.

A gumma develops much more slowly than the multiple sarcomas of the skin, which are frequently bluish in color, as they often contain pigment (melano-sarcoma), and the acute painful nodules, accompanied by fever, which develop in erythema nodosum. It may be very difficult to differentiate between a non-ulcerated gumma and the tumors which develop in mycosis fungoides.

A solitary syphilitic ulcer may resemble very closely a carcinoma of the skin. In a syphilitic lesion there will be no enlargement of the adjacent lymph nodes unless there is secondary infection, and even then the lymph nodes will not be indurated as they are when infiltrated with carcinoma cells. The borders of the syphilitic ulcer are not indurated and craterlike as the borders of the carcinomatous ulcer are. The floor of a carcinomatous ulcer, from which comedolike masses may be squeezed

out, is indurated and fissured and bleeds easily, while the floor of a syphilitic ulcer is less hard, contains caseous, necrotic tissue, and bleeds only where healthy granulations have developed.

Demonstration of the bacilli of glanders by microscopic examination or animal experiments, and the inefficiency of anti-syphilitic treatment prevent confusion with chronic glanders. The demonstration of bacilli in the nodules and ulcers of leprosy and a number of other characteristics guard against mistakes in diagnosis in countries where both leprosy and syphilis occur. Actinomycosis of the skin is rare, and the ulcers developing in this disease resemble those of tuberculosis. From the fistulæ which form the sulphur yellow bodies, composed of colonies of ray fungi, are discharged.

Blastomycetic dermatitis may be mistaken for a syphilitic skin lesion (see p. 391).

In all doubtful cases an energetic anti-syphilitic treatment should be instituted. If there is no improvement after two weeks, the lesion is not of a syphilitic nature. If there is a question as to malignancy, operative removal should no longer be postponed after a trial treatment of this duration.

Treatment.—The gummata and ulcers heal rapidly when early and energetic anti-syphilitic treatment is instituted. The local treatment consists of the application of blue or white precipitate ointment, curetting of the ulcers, and incision and curettage of the softened nodule. Repair is often hastened by local treatment. Plastic operations are often indicated to correct the deformities resulting from the contraction of scars upon the face.

(b) SYPHILIS OF MUCOUS MEMBRANES

Besides the primary lesion, which occurs most frequently as an indurated ulcer upon the margin of the lips, the tonsils, the gums, and the tongue, and can scarcely be mistaken for any other lesion, because of its rapid development and the early and extensive involvement of adjacent lymph nodes, there are a number of lesions of the mucous membranes (especially of the mucous membrane of the mouth cavity) which develop in the second stage of the disease. These develop at the same time as the eruption or recur independently. Sharply delimited erythematous areas, round and flat, pearl gray or bluish white papules, and suppurating ulcers and rhagades, the floors of which are covered with necrotic, caseous tissue, develop upon the buccal mucous membrane during this stage. They correspond to the macules, papules, and ulcers which develop in the skin.

Rhagades develop most frequently about the angles of the mouth,

papules upon the tongue, and the lesions which are known as syphilitic angina upon the soft palate and in the pharynx. In syphilitic angina the uvula and the pillars of the fauces become reddened and congested. The hyperæmia is semicircular in outline, and is sharply delimited from the healthy mucous membrane anteriorly, and can be easily differentiated in this way from the indistinct redness of the ordinary angina, which gradually fuses with that of healthy mucous membranes. Sometimes the tonsils become swollen, painful, and covered with a gray membrane or ulcerated, the lesions resembling somewhat those occurring in faucial diphtheria. In syphilis, however, there is no fever and inspection will reveal grayish white, opalescent papules (plaques opalines) upon the soft palate and the posterior wall of the pharynx and other lesions of syphilis.

Gummatous lesions develop most frequently in the mucous membranes of the mouth, nose, pharynx, larynx, and rectum. The process may extend deeper, involving cartilage and bone and producing extensive destruction of the tissues involved. Perforation of the soft and hard palate, of the septum and bridge of the nose, and cicatricial stenosis of the larynx (following gummatous ulcers upon the arytenoid cartilages, in the true and false vocal cords) and of the rectum are frequent. Disease of the nasal mucous membrane is indicated by a foul-smelling, purulent discharge (*ozæna*); then the bridge of the nose gradually sinks in, as its bony framework is destroyed by the syphilitic osteitis. If this destructive process extends from the nose or pharynx to the base of the skull, a fatal meningitis may develop.

The diagnosis of a lesion resulting from ulceration of gummata of the mucous membranes is not difficult when the disease is well advanced. The tumorlike gummata developing in the muscles of the tongue, which frequently ulcerate but little, may be mistaken for carcinoma, but the induration and swelling of adjacent lymph nodes (the rule in carcinoma) do not occur with gummata. In doubtful cases a small piece of the diseased tissue should be removed and a microscopic examination made. Frequently the other mistake is made: a carcinoma, in spite of the metastases into lymph nodes, is regarded as a gumma, and anti-syphilitic treatment is continued until the carcinomatous infiltration has become so extensive that operative removal of the disease is out of the question.

Tuberculous ulcers of the mucous membranes are flatter than the gummatous and their borders are not sharply cut, but are irregular and ragged, and the floor of the ulcer contains apple-jellylike granulations.

General anti-syphilitic treatment should be instituted, and a nasal douche or a mouth-wash, depending upon the position of the lesion,

of some mild antiseptic solution (potassium permanganate, acetate of aluminum, boric acid, etc.) should be used. Rapid healing follows a course of treatment with iodid of potassium, if secondary infection of the specific osteitis has not already developed. Then it may be impossible to prevent perforation of the palate, destruction of the nasal bridge, etc.

(c) SYPHILIS OF MUSCLE

Rheumatic muscle pains, the exact nature of which is not known, may occur in the earliest stages of syphilis, especially in the eruptive stage. Not infrequently muscle contractures develop in the first year after the infection. The biceps brachii and other flexors, occasionally the triceps brachii, are involved. The contractures develop very slowly, sometimes with little, sometimes with severe pain, and may disappear spontaneously. They are usually the result of a fibrous or a gummatous myositis.

In the diffuse syphilitic or fibrous myositis which develops some years after infection, usually one, more rarely many, muscles are involved. Those most frequently involved are the masseter, the muscles of the calf and arm, the sterno-cleido-mastoid, and the sphincter ani externus. The entire muscle swells acutely, sometimes more chronically, as a result of the interstitial inflammation, and becomes hard and painful upon pressure and movement. As a result contractures develop (inflammatory lockjaw, wryneck, etc.) and normal muscular movements are interfered with.

According to Honsell and O. Busse, this form of myositis does not differ histologically from any other form of interstitial myositis. In recent cases, healing with restoration of function follows anti-syphilitic treatment continued for some weeks. In other cases the muscle fibers degenerate and the entire muscle becomes shortened and transformed into a thin, connective-tissue strand. This happens most frequently when small gummata develop in association with the diffuse infiltration (cicatricial gummata).

The muscle gummata belong almost exclusively to the late forms of syphilis, developing from ten to thirty years after the infection.

Circumscribed, firm nodules, which may reach considerable size, develop in the belly of the muscle or near its origin or insertion. Sometimes one, sometimes many nodules develop in a muscle. Not infrequently a number of different muscles are involved simultaneously, sometimes symmetrical muscles. These nodules develop most frequently in the sterno-cleido-mastoid, in the muscles of mastication, in those about the shoulder, of the upper arm, the thigh, the calf, and the gluteal region. Gummata in the muscles of the tongue are frequent.

When it has reached a certain size (as a rule, that of a hazelnut) a nodule which may have given rise to no symptoms is accidentally discovered. Often it is first noted after an injury, and in some cases probably a trauma determines the development of the lesion. A large, single, circumscribed gumma can be palpated much more easily than multiple small gummata situated close together or in an extensively inflamed muscle. They feel larger in contracted than relaxed muscles and move with the muscle until they degenerate and contract adhesions with neighboring structures or discharge externally. If the nodules do not disappear after some time, leaving a scar in the muscle which is adherent to the skin, large abscesses which may rupture easily or deep ulcers with large recesses develop. Both may cicatrize, but there is always a destruction of a large part of the muscle.

The diagnosis of a gumma in a muscle which has not ruptured externally is always difficult unless the nodule is situated in some muscle commonly affected, such as the sterno-cleido-mastoid or muscles of the tongue. Small nodules may be confused with cysticercus or, occurring in the abdominal muscles, with desmoids. Softened, rapidly growing gummata, attached to surrounding structures (bone, blood vessels, skin), may be regarded as soft, rapidly growing sarcomata. Nodules which go on to abscess formation resemble clinically primary or secondary tuberculosis of muscle (especially that secondary to tuberculosis of the chest). Pus discharged from syphilitic lesions is often steel blue in color and thick (von Bramann); frequently, however, it cannot be distinguished from ordinary tuberculous pus.

The diffuse, boardlike infiltration of the muscles of mastication resembles that occurring in facial actinomycosis. An impulse may be transmitted to a gummatous abscess lying over a large vessel and an aneurysm may be simulated. An ulcerated gumma of the tongue may be mistaken for a superficial, less rapidly growing carcinoma. The syphilitic nature of such a lesion may be recognized, even when there are no other symptoms of the disease, by the absence of the characteristics of carcinoma, the fact that the lesion is composed of many nodules, its painless development, and the diminution in size under anti-syphilitic treatment.

A complete but slow absorption of diffuse syphilitic muscle infiltrations and gummata follows energetic anti-syphilitic treatment. In doubtful cases in which it is possible that a malignant growth exists, iodid of potassium should not be continued until the growth becomes inoperable. If there is no diminution in the size of the lesion after two weeks, an operation is indicated. It is often impossible to differentiate between a gumma and a sarcoma even after they are incised, as they are very similar in appearance. Even a microscopic diagnosis may be difficult.

Cuneiform excision hastens the healing of large gummata. Ulcers resulting from the degeneration of gummata should be dressed aseptically. Dressings of mercury ointment may be used to advantage.

(d) SYPHILIS OF THE LYMPHATIC VESSELS AND NODES AND OF THE BLOOD VESSELS

Syphilis of the lymphatic vessels is of little surgical importance. Frequently red streaks develop in the skin covering the dorsum of the penis, secondary to the initial sclerosis, and in the skin of the forearm or about the angle of the jaw when the chancre is extra-genital. The enlarged and indurated lymphatic vessels, which gradually subside, may be palpated beneath these streaks.

The rare gummatous lymphadenitis is the most important syphilitic lesion of lymph nodes, as it is frequently mistaken for other pathologic conditions. The painless swelling of the lymph nodes adjacent to the primary lesion which develops in from one to three weeks after the chancre, and the general enlargement of the nodes beginning with the general infection in the eruptive stage are very characteristic. The nodes are painless, as large as a hazelnut or cherry, never larger than a bean, move freely under the skin and do not suppurate unless there is a secondary infection. The enlargement of the nodes may persist for years.

In gummatous lymphadenitis there develop slowly, most frequently in the submaxillary and inguinal regions, hard, nodular masses which may become as large as a hen's egg. These may be mistaken for neoplasms, for example, of the submaxillary gland; for hard, tuberculous glands, if they become adherent to surrounding structures, soften and discharge externally; for malignant growths, actinomycosis or tuberculous abscesses. Only when deep ulcers form do the unmistakable signs of the specific nature of the disease become pronounced. If the destruction of tissue is extensive, fatal hæmorrhages may follow the erosion of large arteries (innominate vein, femoral artery) (von Esmarch). The enlarged nodes gradually subside after an energetic antisyphilitic treatment. Healing may be hastened by the extirpation of degenerated glands.

A proliferation and cellular infiltration of the walls of the arteries with a tendency to obliteration of the lumina (arteritis syphilitica obliterans) may develop in the late stages of the disease. Smaller vessels —for example, the basilar artery—may be transformed into a fibrous cord. These changes are not associated with gummata or syphilitic nodules in the vessel wall.

Although these arterial changes are not of a specific nature, they

may be differentiated from other forms of arterio-sclerosis. [" The process differs from simple atheroma (1) in attacking small arteries, (2) in affecting the whole circumference of the vessel and not merely patches, (3) the newly formed tissue becomes vascular and does not undergo fatty degeneration, and (4) it leads to narrowing or occlusion of the vessel rather than to weakening and dilatation."—Rose and Carless, " Manual of Surgery," p. 300.]

a *b*

FIG. 197.—SYPHILITIC HYPEROSTOSIS OF THE TIBIA. *a*, Roughened surface of the bone covered with osteophytes; *b*, longitudinal section of the bone. Medullary cavity obliterated. In the center osteosclerosis, above and below osteoporosis.

The changes occur frequently in the terminal branches of the internal carotid artery. Different forms of paralysis may follow the occlusion of these vessels.

Similar changes are found in the arteries and veins which supply a gumma, and regressive changes in a gumma follow the obliteration of these vessels. Only rarely do gummata develop in the arterial wall.

(e) SYPHILIS OF BONE

The gumma is the most important specific lesion of bone. It develops in the periosteum or medulla or extends to the bone from adjacent tissues. The gumma is one of the late lesions of syphilis, but may develop during the eruptive stage of the severer forms of the disease. In the early period a mild form may attack the periosteum only. It has not been demonstrated that trauma is a predisposing factor in the development of an osteal gumma, although it is often claimed to be an etiological factor.

Syphilitic Osteosclerosis and Osteoporosis.—During the development, involution, or suppuration of an osteal gumma, two pathological proc-

Fig. 198.—Syphilitic Periostitis of the Radius. Congenital Syphilis. Boy seven years of age. (From Professor Bevan's Surgical Clinic.)

esses, *destructive* and *constructive*, are always combined. The granulation tissue of the gumma infiltrates the bone, causing caries or necrosis, and at the same time the surrounding tissue is stimulated to the production of new bone. The surface of the diseased bone, therefore, appears irregular, roughened, and eroded, but the bone as a whole may be larger than is normally the case. Osteophytes and partial hyperostoses develop as the result of this new bone formation. If the entire

bone is involved, it may become hardened and thickened (osteosclerosis, eburnatio), or, as the result of the excessive lacunar absorption, it may become rarefied (osteoporosis); fractures follow this abnormal fragility (osteopsathyreosis).

Syphilitic Periostitis.—In both congenital and acquired syphilis, periostitis frequently develops during the eruptive stage. It develops simultaneously in many different bones, most frequently upon the frontal and parietal, the tibia, sternum, and clavicle. A flat, elastic nodule, the size and shape of an hour-glass and covered by normal skin, develops upon the surface of the bone involved. Only when such a nodule attains considerable size does the skin covering it become reddened and œdematous.

Macroscopic Appearance of a Gumma of Bone.—Incisions made in gummata under a wrong diagnosis have revealed a thick, tenacious fluid; for in this form of gummata a gelatinous tissue develops in the inner layers of the periosteum, from which processes extend into the dilated Haversian canals. According to von Bergmann, the periostitis developing in the earlier stages of syphilis cannot be differentiated from the periosteal gumma, and the former is to be regarded as a mild form of the latter. The small infiltrations disappear rapidly without ulcerating or suppurating, leaving but little trace of previous involvement of the bone. A slight depression in the surface of the bone, the result of a caries sicca, may remain for some time and indicate the location of the previous lesion.

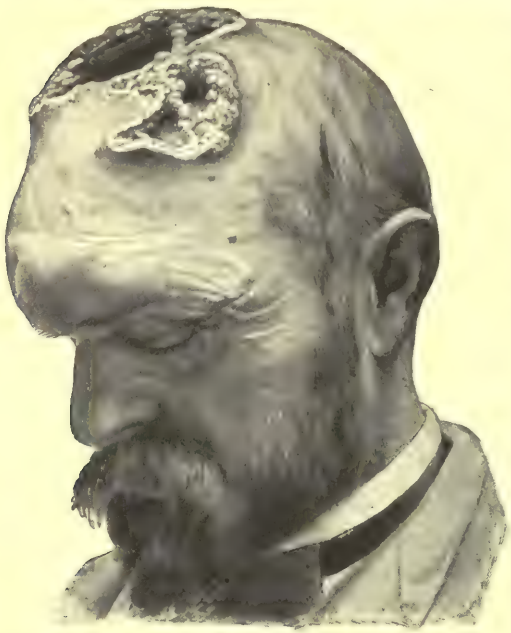


FIG. 199.—LARGE SYPHILITIC ULCERS OF THE HEAD WHICH FOLLOWED THE BREAKING DOWN OF PERIOSTEAL GUMMATA INVOLVING THE FRONTAL AND PARIETAL BONES.

Gummatous Periostitis Developing in the Late Stage of Syphilis.—The gummatous periostitis developing in the late stage of syphilis is characterized by its slow growth, its chronicity, and the size which the lesions attain. The gumma develops in the inner layers of the periosteum and passes along the blood vessels of the Haversian system to

penetrate the bone. Flat, circumscribed, only slightly painful nodules (called tophi) develop simultaneously in different parts, most frequently upon the frontal and parietal bones, the ribs, sternum, clavicle, and bones of the forearm. These nodules vary in size from that of a dollar to that of a man's fist, and in the beginning have an elastic feeling and are covered by normal skin. They soon, however, undergo a number of regressive changes. After a caseous or fatty degeneration of the tissue composing it, the gumma either becomes absorbed or suppurates. If the nodule becomes absorbed, its center gradually softens and sinks

FIG. 200.—GUMMATOUS PERIOSTITIS AND OSTEITIS WITH NECROSIS AND A DEFECT IN THE SKULL.

until finally the entire mass disappears completely. A depression surrounded by a wall-like, irregular border, the result of the ossifying periostitis, remains after a gumma is absorbed. If suppuration occurs, fluctuation, indicative of softening, becomes much more distinct and the skin covering the lesion becomes thinned and reddened. If the liquefied mass is not removed by aspiration or incision, the skin breaks down and a large ulcer forms. Large ulcers which discharge a mucoid, often foul-smelling, pus in which may be found caseous particles and necrotic strands of tissue follow the regressive changes in these lesions. The destructive lesion gradually extends until the surface of the bone is bared and denuded; even dead bone may be felt in the floor of the ulcer. The borders of such a lesion are separated from the subjacent tissues

by the yellow cicatricial tissues of the gumma. A thin layer of bone, corresponding in size to the area involved in the gummatous osteitis, becomes necrotic. Round, disklike, often perforated pieces of sclerotic bone gradually become separated from the thickened surrounding bone. Repair begins with the formation of healthy granulation tissue, and is completed by the formation of a scar, which becomes firmly attached to the underlying bone. The surface of the bone is changed; it is thickened and contains nodules and depressions. Where many periosteal gummata develop at the same time and extend deeply into the underlying bone, as is frequently the case in the skull, hyperostoses of considerable size remain after the lesions heal.

Gummatous Osteitis.—Gummatous osteitis is frequently associated with periostitis, being secondary to it. If suppuration accompanies this form of osteitis, as frequently occurs when the hard palate, the nasal and facial bones are involved, the bone may be completely destroyed. Perforation of the palate and syphilitic saddle-nose, resulting from destruction of the bony framework and subsequent sinking in of the bridge of the nose, are the most common and well-known examples of this form of osteitis (Fig. 201). The only nasal deformity that resembles this at all is the traumatic saddle-nose which follows infected compound fractures of the nasal bones. Osteal gummata and osteophytes developing upon the inner surface of the skull bones or growing into the orbit may exert pressure upon the cortex of the brain or the optic nerve and prove dangerous in this way.

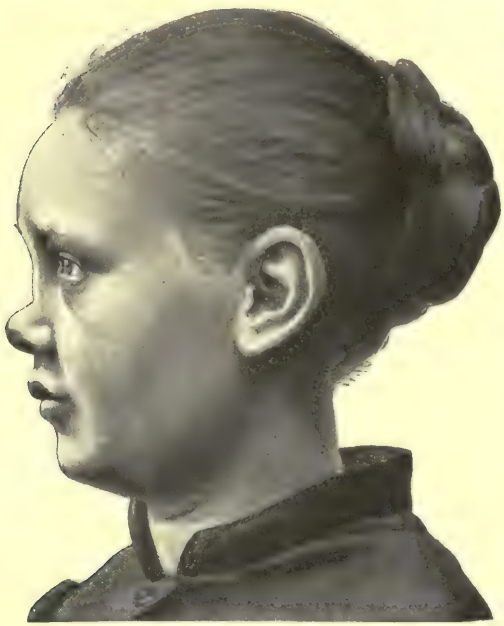


Fig. 201.—Syphilitic Saddle-nose.

The surface of bone is frequently involved secondarily by gummata of the skin and soft tissues. The frontal, parietal, and facial bones are often destroyed by this chronic inflammatory process or transformed into an unsightly, shapeless mass of bone.

Gummatous Osteomyelitis.—In gummatous osteomyelitis, which is rarer than gummatous osteitis, grayish red, gelatinous foci, varying in

size from a pea to a nut, develop in the marrow of bone and in the medullary spaces of the spongiosa and diploë. These foci, which sometimes cause no symptoms, at other times excruciating pain (dolores osteoscopi), later undergo fatty changes and become yellowish and friable.

The bone infiltrated by these foci, which are frequently multiple, gradually liquefies, while the bone surrounding them becomes thickened and sclerotic. If many foci coalesce, small or larger sections of the bone may be deprived of their nourishment and become necrotic. A syphilitic sequestrum separates slowly, frequently lying bare in a wound for years without being completely separated from the surrounding involucrum. The slow separation —that is, formation of the line of demarcation—is due to the slight vascularity and the sclerosis of the surrounding bone.

FIG. 202.—DEFECT IN THE SKULL CAUSED BY A GUMMATOUS OSTEOMYELITIS. (After Heineke.)

The frontal and parietal bones are most frequently involved in this form of the disease, the lesion beginning either upon the inner or outer surface of the bone and gradually extending through it. The macerated skull has, therefore, in spite of large and extensive hyperostoses, a perforated, wormeaten appearance. If suppuration occurs, the clinical picture resembles that following suppuration and rupture of a periosteal gumma.

When repair occurs, the irregular, ragged, disklike sequestra, which sometimes may be very large, are slowly separated from the sclerotic bone surrounding them. Large defects, which may extend to the dura, result from the separation of such sequestra.

FIG. 203.—CONGENITAL SYPHILITIC DACTYLITIS.

Syphilitic Dactylitis.—Short, hollow bones, when a gumma develops within their interior, become expanded and thickened as the result of the growth of the gumma and the accompanying periostitis. Involvement of the metacarpal and metatarsal bones and phalanges gives rise to the clinical picture of spina ventosa. Not infrequently the same flasklike ex-

pansion occurs as in tuberculosis. This form of the disease, called syphilitic dactylitis by Lücke, may be primary or secondary to gummatous inflammation of the surrounding soft tissues. Ulcers and fistulæ may result from regressive changes in the gummata, and entire necrotic phalanges may be extruded or absorbed without accompanying suppuration. These changes occur in both acquired and congenital syphilis. In the latter the lesions are frequently multiple and do not rupture externally (Hochsinger).

FIG. 204.—PATHOLOGICAL FRACTURE OF THE SHAFT OF THE RADIUS. Specimen removed from a man fifty-six years of age, who had suffered from syphilis for sixteen years. Fracture occurred while the patient was supporting himself upon the arm while turning over in bed. A doughy, fusiform expansion of the bone could be palpated. The skin covering the diseased bone was perfectly normal. (From Volkmann's "Diseases of the Organs of Locomotion.")

Syphilitic caries of the vertebræ is rare. If this develops in the upper part of the spinal column, sudden and dangerous falling together of the bodies of the vertebræ may occur.

The spongy articular ends of the diaphysis are but rarely involved. They may become expanded, when diseased, to resemble a tumor. The ligaments and cartilages are destroyed when the process ruptures into the joint cavity. It is sometimes difficult to differentiate this lesion from a central sarcoma or a chronic suppurative osteomyelitis.

The diaphyses of long, hollow bones become diseased more frequently than the articular ends, the bones of the leg and forearm being most commonly attacked. The central gumma produces a slowly developing fusiform expansion of the bone. The bone is infiltrated by the granulation tissue of the gumma and becomes porous, and its resistance is so reduced that pathological fractures may occur, notwithstanding the fact that the bone surrounding such a lesion becomes thickened and sclerotic (von Volkmann, Fig. 204).

Diffuse Syphilitic Periostitis and Gummatous Osteomyelitis.—Besides the localized gummatous periostitis and osteomyelitis there is also a diffuse form. This form of the disease, usually running its course with suppuration, may cause extensive destruction of the bones of the skull; in the long bones, especially in those of the forearm and leg, it may produce large hyperostoses. The bone involved may gradually become thickened,

sclerotic, and heavier than normal, or as a result of the osteoporosis more fragile and lighter.

If the gummatous inflammation occurs in early childhood (the disease being acquired early or being congenital) a characteristic deform-

FIG. 205.—SYPHILITIC OSTEITIS DEFORMANS (THIRTY YEAR OLD MALE PATIENT) WITH ROENTGEN-RAY PICTURE.

ity develops in the bones of one or both legs. This form of syphilis of bones was first described by Fournier as *osteitis deformans syphilitica*. The tibia becomes lengthened, curved forward, and thickened, and a prominence develops upon the anterior surface which becomes more prominent than the calf of the leg. The "saber-sheath" deformity of the tibia is due to a thickening and lengthening of the bone

as a result of the inflammation. The curving of the tibia forward is not to be regarded as static and compensatory (Schuchardt), but is due to the lack of corresponding increase in the length of the fibula and to the traction exerted by the muscles attached to the latter (Weiting). A similar deformity may occur in rickets, but in this disease the epiphysis will be involved and rachitic changes will be present in other bones (Fig. 205).

Osteochondritis Syphilitica.—In congenital syphilis characteristic lesions, called by Wegner SYPHILITIC OSTEOCHONDRITIS, are frequently found in the epiphyses. The epiphyses of the newborn become enlarged, the enlargements being painless. Sometimes the epiphyses become separated, and as a result the diseased limb may appear to be paralyzed (pseudo-paralysis). The process may also extend to the neighboring joint.

The pathological changes are most marked in the epiphyses, but not infrequently they extend to the shaft of the bone, differing in this way from rickets. The calcified cartilage nearest the diaphyses becomes friable and opaque and the epiphyseal line becomes widened, irregular, and wavy. The adjacent medullary cavity contains a grayish yellow, translucent, granulation tissue which has undergone fatty degeneration. Separation of the epiphysis may follow the development of this tissue. Gummata may at the same time develop in the medulla and in the inner layer of the periosteum. The interference with endochondral bone formation may be indicated by shortening or lengthening of the bone.

Syphilitic Osteopsathyreosis.—Fragility of the bones (osteopsathyreosis) occurs in the old, severe cases of syphilis, as in other chronic infectious diseases. It is a result of the cachexia. According to Charpy, there is usually in these cases a considerable reduction of the calcium fluorid in the bones.

Diagnosis of Syphilis of the Bone.—The diagnosis of syphilitic disease of bone is not difficult when the disease is well advanced and the lesions develop in bones which are frequently affected. On the other hand, the diagnosis may be very difficult when there are no other symptoms of the disease. Deep-lying, gouty tophi, or tuberculous abscesses attached to the bones of the skull, forearm, hands, and leg resemble very closely periosteal gummata, provided these have not ruptured. When syphilis produces an enlargement and expansion of a section of a bone, a diagnosis of chronic suppurative or tuberculous osteomyelitis, or of a central or periosteal sarcoma may be made. Multiple hyperostoses also develop in the sclerotizing form of suppurative osteomyelitis. In Paget's disease (*osteitis deformans*) the bones become expanded and deformed. In doubtful cases in which the clinical course

gives no clue to the nature of the disease, anti-syphilitic treatment should be instituted. Roentgen-ray pictures reveal nothing characteristic.

Treatment.—Besides the general treatment which is effective in the early stages of the disease, surgical measures are often required. Painful and ulcerated foci should be exposed, if necessary, with a chisel, and if possible the gummatous masses should be removed with a sharp spoon; sequestra should be extracted. Gummatous abscesses, if small, should be aspirated; if large, they should be incised, curetted, and treated by the open method.

Defects in the skull following removal of sequestra may be closed most quickly by an osteoplastic operation. Extensive defects may be reduced much in size by the gradual regeneration of bone (Hofmeister).

(f) SYPHILIS OF THE JOINTS

In the course of syphilis, one or many joints may become inflamed and the arthritis may pursue different clinical courses. The knee and elbow joints are most frequently involved.

In acquired syphilis a painful arthritis with serous exudate, resembling acute articular rheumatism, may develop during the eruptive stage, likewise during the relapses. This may subside after some weeks if the extremities are immobilized and general treatment is instituted. The restoration of function may be complete.

Syphilitic Hydrops.—A chronic, resistant hydrops, rarely ending in suppuration, may develop late in syphilis. It follows the development of gummata in the synovial membrane and the articular cartilages. The knee is most commonly attacked, and disease is often symmetrical in its distribution. There is but little interference with motion and but slight pain. The diseased synovial membrane becomes thickened and covered with villous, even tumorlike, masses. The articular cartilage becomes eroded, the pathological changes being more marked in the center than at the edges, and small but deep cicatricial defects form which often radiate, like other syphilitic scars (chondritis syphilitica, Virchow).

The diagnosis is frequently difficult, especially when there are no characteristic symptoms or signs of syphilis. The involvement of many joints and the relatively little disturbance of function should suggest syphilis. The treatment, in addition to the general treatment, consists of removal of the exudate and the application of a compression dressing. Frequently, after a long time, the healing is complete. Not infrequently the capsule is thickened and the articular cartilages are partly destroyed. If this is the case, there will be some limitation of motion

even when healing is complete. A grating sensation, elicited when the bones entering into the formation of the joints are moved, is indicative of the destruction of the articular cartilages. Palpable, tumorlike growths in the capsule have been successfully extirpated (Borchard).

Arthritis Following Rupture of an Intraosteal or Periosteal Gumma.—The arthritis which is secondary to the rupture of an intraosteal or periosteal focus into the joint cavity pursues the severest clinical course. All of the ligaments and the articular cartilages may be destroyed. Flail joints or anchylosis with contractures develop most frequently in the fingers and toes, the soft tissues, bones and joints of which may all be involved (dactylitis syphilitica).

Acute Gummatous Arthritis.—Schuchardt has shown that there is also an acute gummatous arthritis. Operations have been performed upon cases in which a diagnosis of gonorrheal arthritis had been made, and miliary gummata have been demonstrated in the tissues removed from the thickened capsule.

In children with congenital syphilis, not infrequently an exudate accompanied by but few symptoms develops rapidly in a number of joints (most frequently in the knee and elbow). This form of arthritis is secondary to a syphilitic osteochondritis or to a gummatous inflammation of the epiphysis and synovial membrane. Suppuration in this form of arthritis is rare. The diagnosis is difficult, unless there are other lesions of syphilis, such as an interstitial keratitis. Under general treatment the arthritis subsides without any limitation of motion. Operative interference is indicated only when fistulæ persist or the joint suppurates.

(g) SYPHILIS OF THE TENDON-SHEATHS AND BURSÆ

An acute exudative inflammation of tendon-sheaths and bursæ may develop during the eruptive stage of syphilis. It is comparable to the serous arthritis occurring during this period, and like it usually subsides rapidly. Frequently gummata develop in bursæ, especially in those about the knee joint. They develop slowly and without pain, and may rupture through the skin or through the capsule of the joint. A gumma in the skin or bone may extend to a bursa, the latter becoming secondarily involved. Syphilis of the tendon-sheaths is most frequently secondary to syphilis of bone—for example, in syphilitic dactylitis.

The diagnosis of the acute exudative tendo-vaginitis and bursitis occurring in the eruptive stage is not difficult. The diagnosis of the gummatous form is difficult, especially if the lesion has not perforated the skin, if there are no ulcers or other lesions characteristic of the disease.

The treatment consists of exposure and removal of the gummatous masses, if the lesion does not rapidly improve under general anti-syphilitic treatment.

(h) SYPHILIS OF THE DIFFERENT VISCERA

Brief mention will be made of the syphilitic lesions of the **brain, liver,** and **testicle,** as these are the most important to the surgeon.

Large gummata developing within the brain substance produce the symptoms of brain tumor. The same symptoms may be produced by gummatous masses and exostoses developing upon the internal table of the skull bones and producing pressure. Syphilitic changes in the cerebral arteries give rise to transitory and permanent functional disturbances, the permanent disturbances being always associated with infarction and subsequent softening of brain tissue. Apoplexy occurring in early life is frequently due to the arterio-sclerosis associated with syphilis.

General interstitial syphilitic hepatitis is of less interest to the surgeon than the hepatitis associated with the development of nodular, gummatous masses. Sometimes it is difficult to diagnose between syphilis and carcinoma of the liver. The masses occurring in syphilis are not so hard as those found in carcinoma, and in syphilis notches develop in the edge of the liver as the result of the contraction of the interstitial tissue lying between the gummata.

Syphilis of the testicle occurs in two forms, as a diffuse interstitial and a gummatous orchitis. It is often difficult to differentiate the gummatous masses, before the skin has become involved and the typical ulcers have formed, from the hard, nodular masses developing in malignant growths. Involvement of the regional lymph nodes speaks for malignancy.

In regard to the **prognosis** of syphilis it should be mentioned that the majority of the lesions which the surgeon sees belong to the late forms, and these develop only in severe cases or in cases which have been improperly treated. It should also be remembered that there are probably other severe organic lesions when gummata develop, even if the latter subside rapidly under anti-syphilitic treatment.

Treatment.—Preparations of mercury and potassium iodid are employed in the treatment of syphilis. Good nutritious food and a hygienic mode of life are very essential.

The most reliable preparation of mercury is the blue or gray ointment (Unguentum hydrargyri cinereum). From ʒij to iij of the ointment should be rubbed in daily into different parts of the body. In children from gr. 15 to 45 should be used. The entire surface of the trunk and extremities should be gone over in six days. A full bath

should be given on the seventh day and the ointment applied again for six days, being rubbed into the skin of the trunk and extremities as before.

Directions should be given the patient regarding the care of the teeth, and a good mouth wash (water to which are added a few drops of tincture of myrrh or acetate of aluminum) should be prescribed in order to prevent mercurial stomatitis with the accompanying inflammation and ulceration of the gums, loosening of the teeth, *fœtor ex ore*, and salivation. If the stomatitis becomes severe, the inunctions should be stopped and the ulcers cauterized.

Subcutaneous injections and internal administration of mercurial compounds are less reliable than the treatment by inunctions. Although there is less danger of mercurial poisoning and administration is simpler, when mercury is injected subcutaneously or administered internally recurrences are more frequent. Mercurial treatment should be begun with the development of the eruption, and during the following three years' treatment should be continued at intervals, even if the symptoms of the disease do not recur (Fournier and Neisser).

Potassium iodid is usually employed in the treatment of later syphilitic lesions (a saturated solution of potassium iodid in water being employed). It is well to begin with small doses and gradually increase the dosage until results are obtained. In adults it is a good rule to begin with 10 drops three times a day and to increase the dose from 1 to 2 drops each day until symptoms of iodism develop or the lesions or symptoms improve. The drug should be administered after meals, and may be given in milk or a glass of water. In severe cases a mixed treatment of mercury and potassium iodid may be employed. Frequently potassium iodid is used for diagnostic purposes. When used for diagnostic purposes in doubtful cases it should be emphasized that the drug should not be continued longer than two weeks unless there is improvement. If continued longer without any improvement, the doubtful lesion—for example, a carcinoma—may become inoperable.

The symptoms of iodism consist of coryza, conjunctivitis, headache, and acne. In severe cases large furuncles may develop. If the symptoms are severe the iodid preparation should be stopped. Mild symptoms often subside when the preparation is continued.

Calomel is best suited for internal administration in treating syphilitic lesions in small children. Children a few weeks old may be given from $\frac{1}{15}$ to $\frac{1}{10}$ gr.; children three months old, $\frac{1}{100}$ gr.; if older, $\frac{2}{10}$ to $\frac{3}{10}$ gr. three times daily (Lesser).

LITERATURE.—*O. Busse.* Ueber syph. Entzündungen bei quergestreiften Muskeln. Arch. f. klin. Chir., Bd. 69, 1903, p. 485.—*Borchard.* Ueber luetische Gelenkentzündungen. Deutsche Zeitschr. f. Chir., Bd. 61, 1901, p. 110.—*v. Bramann.* Ueber syph.

Geschwülste in den Muskeln. Berl. klin. Wochenschr., 1889, p. 120.—*Doutrelepont* und *Gouven.* Ueber d. Nachw. v. Spiroch. pall. in tertiär syph. Produkten. Deutsche med. Wochenschr., 1906, p. 908.—*v. Esmarch.* Zur Diagnose der Syphilome. Chir.-Kongr. Verhandl., 1895, II, p. 298.—*Fischer.* Krankheiten der Lymphgefässe, Lymphdrüsen und Blutgefässe. Deutsche Chir., Stuttgart, 1901.—*Hochsinger.* Zur Kenntnis der hereditär-syph. Phalangitis der Säuglinge. Festschrift für Kaposi, Wien, Leipzig, 1900;—Studien über die hereditäre Syphilis. Wien, 1904, II. Teil: Knochenerkrankungen.—*Hofmeister.* Ueber die Regeneration der Schädelknochen. Beitr. zur klin. Chir., Bd. 13, 1895, p. 453.—*Hoffmann.* Nachtrag zu der Arbeit von Schaudinn und Hoffmann. Berlin. klin. Wochenschr., 1905, p. 726.—*Honsell.* Diffuse syphilitische Muskelentzündung. Beitr. z. klin. Chir., Bd. 22, 1898, p. 502.—*Kaposi.* Pathol. und Therap. der Syphilis. Deutsche Chirurgie. Stuttgart, 1891.—*Kaufmann.* Pathol. Anatomie, Berlin, 1901.—*Klemm.* Ueber die zentrale gummöse Osteomyelitis der langen Röhrenknochen. v. Volkmann's Sammlung klin. Vortr., N. F., No. 273.—*Lang.* Vorlesungen über Syphilis. Wiesbaden, 1896.—*Lesser.* Geschlechtskrankheiten. Leipzig, 1901.—*Lexer.* Zur Beurteilung des Wertes der verschiedenen Quecksilberpräparate in der Syph.-Therap. Arch. f. Derm. u. Syph., Bd. 21, 1889, p. 715.—*H. Lorenz.* Die Muskelerkrankungen. Wien, 1898.—*Mulzer.* Sammelreferat über Spirochaetenbefunde bei Syphilis. Archiv f. Dermat. u. Syph., Bd. 79, 1906, p. 387.—*A. Neisser.* Versuche zur Uebertragung der Syphilis auf Affen. Deutsche med. Wochenschr., 1906, p. 493.—*Pielicke.* Die syph. Gelenkerkrankungen. Berl. klin. Wochenschr., 1898, p. 78.—*Schaudinn* und *E. Hoffmann.* Vorläufiger Bericht über das Vorkommen von Spirochaeten in syphil. Krankheitsprodukten und bei Papillomen. Arbeiten aus dem kais. Gesundheitsamte, Bd. 22, 1905, p. 2;—Ueber Spir. pall. bei Syph. und die Unterschiede, etc. Berliner klin. Wochenschr., 1905, p. 673.—*Schuchardt.* Die Krankheiten der Knochen und Gelenke. Stuttgart, 1899.—*Virchow.* Die krankhaften Geschwülste, Bd. 2, p. 392.—*Wieting.* Zur Säbelscheidenform der Labia. Beitr. z. klin. Chir., Bd. 30, 1901, p. 615.—Handbuch der prakt. Chirurgie von *v. Bergmann, v. Bruns* und *v. Mikulicz,* I. Bd., *v. Bergmann,* p. 137.

Literature Concerning the Spirochæta Pallida.—*Tomasczewski.* Ueber den Nachweis der Spirochaete pallida bei tertiärer Syphilis. Münchener med. Wochenschr., 3 Juli, 1906, p. 1301. Nachweis von Spirochaeten in offenen und geschlossenen Gummata.—*Simmonds.* Ueber den diagnostischen Wert des Spirochaetennachweises bei Lues congenita. Ibid., p. 1302. Wo Syphilis congenita nicht vorliegt, sind (nach Untersuchungen von 26 Fällen) Spirochaeten in den Organen von Säuglingen und Föten selbst bei vorgeschrittener Mazeration nicht anzutreffen. Dagegen fanden sie sich bei vier mazerierten Föten syphilitischer Herkunft in der Haut, in Muskeln und Knochen und in sämtlichen Organen, ferner sehr reichlich im Mekonium. Bei der syphilitischen Osteochondritis waren sie nur in der Knorpelknochengrenze und in dem benachbarten Perioste erkennbar.

CHAPTER XIII

RHINOSCLEROMA

Rhinoscleroma, described first by Hebra and Kaposi (1870), is a chronic, progressive disease which usually begins in the mucous membrane of the nose, less frequently in that of the pharynx, larynx, or

palate, and then extends to neighboring parts—the nose, tear passages, trachea, and lips. The disease is rare in Germany, very common in Austria and Southwestern Russia; occasionally cases are seen in Central America and Italy. The disease develops most frequently in adults of the middle class.

The inflammatory masses, which are infectious granulomata, resemble those found in tuberculosis, syphilis, actinomycosis, leprosy, and glanders. They develop first in the naso-pharynx and choanæ, and then extend either to the mucous membranes of the nose, lips, cheeks, and gums or to those of the upper respiratory passages. Primary as well as secondary foci may develop in the pharynx, trachea, and larynx.

The disease develops slowly and pursues a very chronic course. The infiltration of the mucous membranes sometimes appears in the form of nodules; at other times in the form of tumors or flat, thickened, firm areas of a cartilaginous consistency. The edges of these infiltrated areas may be sharply defined or may gradually fuse with the surrounding healthy tissue. The skin or mucous membrane covering such lesions is reddened, tense, and traversed by dilated veins. It is firmly attached to the inflammatory mass and may be dry, fissured, or ulcerated. Extensive destruction of the infiltrated areas does not occur; on the other hand, there is a tendency to cicatricial contraction. Cartilage to which the pathological process extends becomes fibrillated or hyperplastic.

Gradually the nasal passages become occluded. These masses developing in the pharynx, larynx, and epiglottis may cause considerable interference with respiration and deglutition. When the skin of the nose is involved it becomes hard and bluish red in color, and the nose proper becomes broad and deformed, the alæ nasi become separated, and the hard inflammatory masses extend to the lips.

All these changes, which eventually lead to cachexia or favor the development of some lesion in the lungs, develop within ten years.

Rhinoscleroma may be confused with sarcoma, carcinoma, and syphilis. In doubtful cases a microscopic examination of a piece of tissue should be made.

In the beginning of the disease the lesions should be extirpated. If necessary, the nose should be split, so that free access may be had to the lesion. As a rule the results following extirpation are not permanent, as the disease tends to recur. If complete removal is impossible, small pieces should be excised in order to reëstablish the nasal passages. If the disease develops in the larynx, tracheotomy may be necessary.

Microscopic examination of the lesions reveals a characteristic con-

nective-tissue proliferation which is most marked about the blood vessels. The cells of the connective tissue undergo a peculiar degeneration, large, swollen, vacuolated cells filled with bacilli, cells which have undergone hyaline degeneration and free hyaline masses being found in these lesions. Large numbers of bacilli lie in the lymphatic spaces or in the cells.

The bacillus demonstrated by von Frisch in 1882 is probably the cause of rhinoscleroma. It is a short, rounded rod, provided with a mucous capsule, frequently two bacilli being found within the capsule (diplo-bacillus). Notwithstanding the fact that this bacillus is found so constantly in the lesions of the rhinoscleroma, it has not been demonstrated that it is the cause of the disease, as animal experiments have been negative and the bacillus resembles closely other bacteria, such as Friedländer's pneumo-bacillus, the bacillus of ozæna, and other encapsulated bacteria occurring in the nose. It is impossible to make an accurate differentiation between the forms mentioned.

LITERATURE.—*Babes.* Das Rhinosklerom. In Kolle-Wassermann's Handb. der pathog. Mikroorganismen, Bd. 3, 1903, p. 408.

CHAPTER XIV

BOTRYOMYCOSIS

ACCORDING to Bollinger, botryomycosis is a disease characterized by the development of infectious granulomata in the skin, grapelike or mulberrylike groups of cocci being found in the tissue. These can hardly be differentiated, even by cultural methods, from the staphylococcus pyogenes aureus. Küttner doubts their specificity. The disease occurs frequently in horses; more rarely in other domestic animals, and only occasionally in man.

The disease was first demonstrated in man by Poncet and Dor in 1897. Small nodular tumors which, as Legrain's illustrations show, may sometimes become as large as a fist, develop in the skin. The surface of such masses, which is often ulcerated and nodular, bleeds easily and profusely. The nodules are soft in consistency, red in color, and are attached to the dermis. These masses grow slowly, without pain, and are usually found upon the fingers, more rarely upon the dorsum of the hand, the arm, and other parts of the body.

These masses can be easily excised, as they do not extend into the deeper tissues. Botryomycosis may be confused with angiosarcoma, which is not infrequently pedunculated and not larger than a cherry

when it develops upon the fingers. The microscopic finding of groups of cocci in the tissue is of diagnostic importance.

LITERATURE.—*Gahinet.* Les tumeurs botryomycosiques chez le cheval et chez l'homme. Paris, 1902, Michalon.—*Glage.* Botryomykose. In Kolle-Wassermann's Handbuch der pathog. Mikroorganismen, Bd. 3, 1903, p. 795.—*Küttner.* Ueber teleangiekt. Granulome. Beitr. z. klin. Chir., Bd. 47, 1905, p. 1.—*Legrain.* Archives de Parasitologie, 1898.

PART III

NECROSIS

The disturbances of nutrition arising in wounds and associated with injuries or diseases of the blood vessels, burns, frost-bites, etc., are often the cause of anxiety to the surgeon. If treatment does not improve the nutrition of the tissue, death of the same may be unavoidable. This condition is known as necrosis or, because of the charred appearance of the tissues, as gangrene. [If death is limited to the soft tissues, the term sloughing or sphacelation is employed, and the dead tissue is spoken of as a sphacelus; gangrene is applied to the necrotic processes which involve both soft tissues and bone.]

Disturbance of nutrition is, as a rule, synonymous with disturbance of circulation of the blood; for the lymph alone, even if its circulation is not interfered with, cannot nourish large areas or parts for any length of time.

A disturbance of circulation follows the cutting and ligation of large blood vessels during operations or in accidental-wounds; the laceration of large vascular trunks in subcutaneous injuries, and their closure by the pressure of blood poured out into the tissues; the narrowing or dilatation of the vessel wall, the result of pathological changes. Whether the circulatory disturbance subsides without permanent injury or causes necrosis or gangrene depends upon a number of factors: (1) whether the circulation is completely stopped or merely interfered with; (2) whether the interference with circulation has been sudden or gradual; (3) whether the cause is removed early or late; and finally (4) whether a collateral circulation can develop rapidly enough to supply the tissues with sufficient blood. This is possible when the anastomosing vessels, which must dilate easily, are present in large number.

General and Local Conditions Favoring Necrosis.—General and local conditions are also very important in determining the fate of the tissues. A poor general condition and cardiac weakness may prevent the recovery of the tissues even after the interference with the circulation has been removed or a collateral circulation has been established. Hæmatomas, inflammatory processes, diseases of the vessels supplying the area,

passive hyperæmia resulting from tight bandages, or the poor position of the limb involved may favor the development of necrosis or prevent *restitutio ad integrum.*

Symptoms of Beginning Circulatory Disturbances.—Threatening circulatory disturbances due to interference with or cessation of the arterial flow are indicated by the symptoms of ischæmia; those due to interference with or cessation of venous return by the symptoms of passive hyperæmia or stasis. The signs of interference with circulation are seen best in a pedunculated skin flap, the pedicle of which is too narrow or too thin or is constricted by the dressing. If the arterial circulation is interfered with the flap becomes pale and cold; its edges, if there is no inflammation, become dry and a brownish black crust develops. If the venous circulation is interfered with the flap swells, sometimes the entire flap becomes blue, and at other times merely blue areas develop. The blood passes from the small arteries into capillaries and veins filled with blood which no longer circulates. The walls of the capillaries and veins are stretched and become permeable as a result of the nutritional disturbances. The blood is poured out into the tissues, and the flap becomes dark blue or black in color when the red blood corpuscles are destroyed and the blood pigment is set free. The serum which raises the epidermis to form the so-called gangrenous blebs is also stained by this pigment. Numerous ecchymoses which develop before the infarction of the flap is complete are due to the bursting of blood vessels. If dark blood is discharged from incisions made into the flap, thrombi have not developed. When thrombosis occurs the tissues die and the fate of the flap is sealed. [When flaps are sutured and there is considerable tension, it is sometimes advisable to make multiple small incisions into the areas which are under the most tension. The venous blood escapes through these incisions, thrombi do not develop, and frequently the flap may be saved.] When the flap is destroyed by the interference with venous circulation, the epidermis gradually becomes separated from the corium and is cast off in shreds and strips; the corium, no longer protected, dries, forming a hard, black mass. Necrosis, beginning at the edge of the flap, gradually extends until the entire flap becomes necrotic, unless proper treatment is instituted.

Disturbances Following Interference with Circulation.—Mild disturbances follow interference with, but not complete cessation of, either the arterial or venous circulation. The resistance of the imperfectly nourished tissue is reduced and chronic eczema develops, the skin atrophies, wounds no longer heal rapidly, and infections when once established persist and extend rapidly. Impaired circulation is an important etiological factor in the development of varicose ulcers and the ulcers developing in arteriosclerosis and diabetes.

Other Causes of Necrosis.—Disturbance of circulation is the most important, but not the only cause of necrosis. Tissues may become necrotic as the result of crushing injuries and the action of chemical and thermal agents. The cell protoplasm, as the result of the action of injurious agents, is no longer able to assimilate foodstuffs, even when enough vessels remain intact to supply the necessary material.

Necrosis and Necrobiosis.—When death of tissues quickly follows the infliction of an injury, it is called *necrosis;* when death follows slowly, and is preceded by degenerative changes in the cells, it is called *necrobiosis.*

Necrosis and Gangrene.—Clinically, dry is differentiated from moist gangrene. *Dry gangrene* (necrosis, mummification) is the result of the occlusion of the artery supplying the area involved, provided a sufficient collateral circulation is not established rapidly. The tissues, previous to their death, are drained of their fluids, or the fluids are lost by evaporation at points where the epithelium is destroyed, and the deeper tissues become dry, shrunken, hard, and black. *Moist gangrene* (necrosis humida) occurs only when the tissues before their death have been œdematous, and in those conditions in which infections, especially with putrefactive bacteria, favor liquefaction of the tissues, a process which is observed in different degrees in every severe suppurative and putrefactive infection. The term gangrene is at the present time applied by many authors to the moist, foul-smelling forms of necrosis only.

Formation of Granulation Tissue and the Line of Demarcation.—Granulation tissue develops about the edges of the dead tissue as the result of a reactive inflammation. It is the same process that occurs in every open wound, about foreign bodies and inflammatory foci. The dead tissue is gradually separated from the living by granulation tissue and the ferments which are liberated by the leucocytes wandering into this zone. The line of demarcation gradually becomes more distinct and the necrotic tissue becomes separated from the slightly elevated wall of granulation tissue by a groove filled with fluid resembling pus.

The necrotic tissue is cast off when enough healthy granulation tissue develops to separate the living from the dead. The time required for the separation of necrotic tissue depends upon the character of the tissue and the size of the necrotic mass; for example, from one to two weeks are required for the separation of necrotic skin, from two to three months for the separation of necrotic bone.

CHAPTER I

NECROSIS DUE TO TRAUMA: (1) THE DIRECT RESULT OF TRAUMA, (2) FOLLOWING INJURIES OF THE BLOOD VESSELS

Pieces of tissue completely separated from neighboring structures become necrotic unless conditions exist or are provided which favor healing. Conditions, however, are rarely favorable, as the tissues are crushed, blood is extravasated, hæmatomas form, and bacterial infections may develop. Healing, as a rule, occurs only when tissues are transplanted, as in these cases conditions are provided which favor rapid healing.

Necrosis Produced by Blunt Force.—Necrosis frequently follows the application of blunt force. The tissues are more apt to become necrotic when acted upon by blunt force, as the vessels are crushed, the intima is lacerated, and thrombi develop, closing the vessels. Machine injuries, in which the tissues are lacerated and the larger blood vessels are torn, may cause in this way necrosis of an entire extremity. Skin which is crushed and separated from the subcutaneous fat and fascia by blunt force frequently becomes necrotic.

Necrosis Following Occlusion of Blood Vessels.—Viscera, an entire extremity or parts of it may become necrotic as the result of an injury, rupture, occlusion (by malignant growths) or ligation (for cure of aneurysm) of the principal artery, providing a sufficient collateral circulation is not established quickly enough to provide for the nourishment of the tissues. General weakness of the patient, pathological changes in the vessel wall, and the pressure of extravasated blood may prevent the establishment of a collateral circulation.

The viscera undergo necrosis most rapidly when the vessels supplying them (arteries or all the veins) are injured or ligated. It is well known that necrosis of the testicle may follow injury of the arteries in the cord in the repair of an inguinal hernia, or in operating upon a varicocele. Necrosis of the kidney, spleen, or intestines follows injury or ligation of the arteries supplying them. [These arteries are terminal, and a collateral circulation is not established even under favorable conditions.] Ligation of the internal carotid artery is followed in about forty per cent of the cases by focal degeneration or necrosis of the brain.

Results of Ligation of Some of the Principal Arteries of the Extremities.—The results following ligation of the principal arteries of the extremities differ. Of course the statistics prepared from operations performed in preantiseptic times are not as good as those prepared

later, when antisepsis and asepsis rendered wound-infections, which often defeated the purpose of the operator, much less frequent.

Gangrene follows in from one to two per cent of the cases of ligation of the common femoral vein (Fränkel, Franz), in about twenty per cent of the cases of ligation of the femoral artery (Raabe), and in from fifty to sixty per cent of the cases of simultaneous division of both the femoral artery and vein (von Bergmann, Ziegler). Gangrene occurs in fifty per cent of the cases of ligation of the popliteal artery, and always follows simultaneous ligation of both artery and vein (von Bergmann, Janssen).

The results following ligation of the arteries of the upper extremity are more favorable. Gangrene follows in only about two per cent of the cases of ligation of the subclavian artery (von Bergmann). Even simultaneous ligation of both the artery and vein is not so dangerous, as the conditions are favorable for the establishment of a collateral circulation. Ligation of the axillary artery is followed by gangrene in six per cent of the cases, of the brachial artery in eighteen per cent (cf. Hoepfner).

Factors Interfering with or Preventing the Development of a Collateral Circulation.—It may be seen from the above that in many cases gangrene does not follow occlusion of the principal arteries and veins, even the simultaneous occlusion of both. Gangrene, however, invariably follows simultaneous ligation of both the popliteal artery and vein. If gangrene occurs, when the anatomic conditions are favorable for the establishment of a collateral circulation, there is, as a rule, some pathological change which interferes with the nutrition of the tissue. The following are the most frequent conditions which prevent the establishment of a collateral circulation: (1) general and cardiac weakness; (2) loss of blood following operation or accident; (3) alterations in the composition of the blood, such as occur in anæmia, diabetes, infectious diseases; (4) arteriosclerosis, dilatation of the veins, thrombosis, inflammation, interference with the circulation by exudation of blood beneath unyielding fascia, by tight bandages, or by the dependent position of the extremity.

Symptoms of Gangrene.—The symptoms which indicate the beginning of gangrene are those of ischæmia or passive hyperæmia. The part of the extremity below the point of occlusion becomes white, cold, and pulseless or œdematous, mottled and dark blue in color, and covered with large vesicles containing a blood-stained serum. The changes are most marked in the distal parts of the extremity. In the beginning there are paræsthesia and dull pain; finally complete sensory and motor paralyses develop unless the condition is relieved. An ischæmic muscle loses its electrical excitability after five hours. Even after complete loss

of sensation a severe, aggravating pain which is referred to the nerves distal to the line of demarcation continues. Motor paralyses develop when the bellies of the muscles have degenerated. Usually the first symptoms of a dry or moist gangrene develop, when the principal vessels are involved, as early as the second day upon the fingers and toes. Unless a collateral circulation is established in a few days the process extends, involving the entire part below the point of occlusion. In favorable cases the gangrene remains limited to the tips of the fingers or toes or to isolated areas in the skin of the hand or foot, which

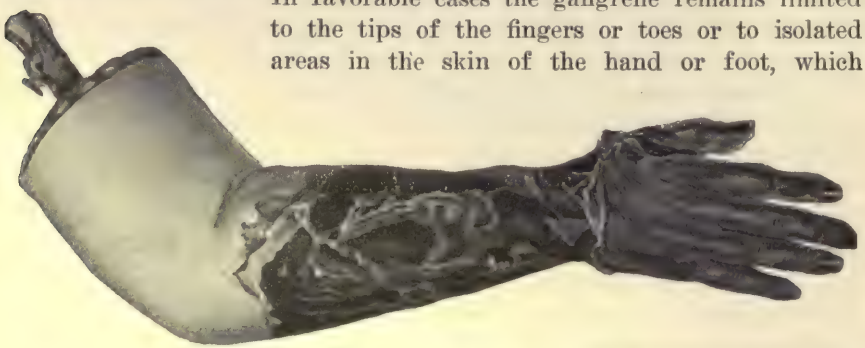


FIG. 206.—NECROSIS (DRY GANGRENE) OF THE ARM FOLLOWING RUPTURE OF A DISEASED AXILLARY ARTERY WHILE ATTEMPTING TO REDUCE AN OLD DISLOCATION OF THE SHOULDER. Patient was a woman sixty-nine years of age. Appearance of the arm four weeks after ligation of the artery.

heal after the necrotic tissue is cast off. The death of the tissue is most rapid when infected. Not infrequently, phlegmonous inflammation and lymphangitis develop as the result of infection of the granulation tissue of the demarcation zone.

Symptoms of Gangrene of the Viscera.—The symptoms of necrosis of the viscera differ, alterations of, interference with or a complete cessation of their specific functions being the most pronounced. The testicle is superficial and presents quite definite symptoms when it becomes necrotic. The scrotum becomes inflamed and œdematous and a serohæmorrhagic exudate is poured out into the tunica vaginalis.

Treatment.—An attempt should be made in the treatment of necrosis, the direct or indirect result of trauma, to prevent the introduction and development of putrefactive bacteria. The surrounding area should be cleansed and sterilized, and a dry, aseptic dressing should then be applied. Only in dry, superficial gangrene of the skin can a moist dressing be applied without danger. In these cases a moist dressing of acetate of aluminum, or boric acid, without rubber protective, hastens the formation of granulation tissue and the separation of the necrotic tissue. Such a dressing should be changed daily or oftener.

Necrotic viscera, such as the spleen, intestine, kidney, and testicle,

should be removed as soon as possible in order to prevent the inflammation of the membrane (peritoneum) and tissues (loose connective tissue of the scrotum) surrounding them.

In gangrene of the extremities the operation should be postponed, unless there are indications for immediate interference, until the line of demarcation becomes well established and distinct. This prevents the unnecessary removal of tissue and the possibility of making the amputation through tissues which will later become necrotic. If lymphangitis, phlegmonous inflammation, or fever accompanied by putrefaction develop, amputation should no longer be postponed. Frequently it is the only procedure which will prevent the development of a general putrefactive infection.

In the after-treatment following ligation of the principal artery of the extremity, an attempt should be made to favor the development of a collateral circulation and to prevent necrosis.

Bandages should be applied loosely, the extremity should be supported upon soft cushions or pillows, hyperæmic areas in the skin should be punctured, and soft tissues infiltrated with large amounts of blood should be incised down to the vessel. If gangrene from venous stasis, the result of the ligation of a vein or of a vein and an artery, threatens, the extremity should be elevated or vertically suspended at once in order to favor the return flow of the venous blood.

CHAPTER II

NECROSIS THE RESULT OF PRESSURE, CONSTRICTION, INVAGINATION, AND TORSION

In this form of necrosis the circulation is interfered with or completely stopped by compression of the vessels.

Decubitus.—*Pressure necrosis* (decubitus) occurs most frequently in feeble, emaciated patients who are compelled to lie in a recumbent or any one particular position for a long time. It develops most frequently over bony prominences, such as the sacrum, the spines of the vertebræ, the spine of the scapula, and the os calcis.

A similar form of necrosis which occurs most frequently upon the lateral margin of the foot, the base of the fifth metatarsal bone, over the heel, the malleoli, the tendo Achillis, the crest of the tibia, the patella, the trochanter major, the spines of the ilium and the chin may follow the use of imperfectly padded or improperly applied splints. More rarely, skin surfaces which are in contact, such as the folds of

the groin, the scrotum, and the inner surface of the thigh, undergo pressure necrosis.

The pain, which is present in the beginning, gradually subsides unless maintained by inflammation. The blue and discolored area exposed to pressure becomes anæsthetic and dries to form a hard, black crust, which is gradually separated from the surrounding structures by granulation tissue. The ulcer which forms when the crust is removed resists treatment, for usually the general condition of the patient is poor and, besides, the ulcer is subjected to continuous pressure. Such ulcers developing over the sacrum frequently become infected, as they are contaminated by urine and fæces. A painful suppurative or putrefactive inflammation which undermines the skin then develops; the fascia becomes gangrenous and the surface of the bone is exposed.

Erysipelas, phlegmons, and severe general infections, associated with metastases which develop from thrombi originating in the inflamed veins of the necrotic area, are frequently terminal events in these cases.

The first indication in the treatment of pressure necrosis is to remove the cause. This is much more easily accomplished when the pressure is produced by a tight bandage than when it follows long confinement in one position. An attempt should be made to prevent bedsores from developing by providing soft, smooth bed clothing, or by using water cushions or rubber rings. The tissues surrounding the area exposed to pressure should be rubbed daily with alcohol, dried, and dressed aseptically. Moist dressings macerate the skin, transfer infection, and favor the development of a pustular eczema. A dressing of ten per cent zinc-vaseline ointment lessens the burning sensation and favors the formation of granulation tissue and cicatrization. The prevention of pressure necrosis is one of the most important duties of a nurse. When a patient has been operated upon and his position cannot be easily changed, especial care should be paid to keeping the bed clothing and the nightgown smooth, and to supplying clean linen. Air or water cushions are appreciated very much by weak, emaciated patients.

The beginner should learn to protect all bony prominences and projecting tendons when applying a plaster-of-Paris dressing.

Necrosis due to constriction may develop when one of the turns of a bandage is too tight or when an Esmarch constrictor, applied to produce an artificial ischæmia, is allowed to remain too long. Ischæmic necrosis may develop when a constrictor is allowed to remain in position for two and a half hours. It is easily understood how much greater the dangers of necrosis are when the constrictor is allowed to remain much longer. Plaster-of-Paris dressings may cause necrosis, especially when applied to an extremity the circulation of which is already interfered with by extravasations of blood or pathological changes in the

vessel wall (arteriosclerosis). Necrosis is preceded, as a rule, in these cases by marked passive hyperæmia, and the tissues are sacrificed to the pressure of the bandages. In all dressings, especially when plaster-of-Paris casts are applied, the fingers and toes should remain free so that after the bandages are applied they may be inspected on the following day. If they are pale or blue, swollen, anæsthetic, or cannot be moved, the dressing should be removed immediately and reapplied with great care, or the limb should be immobilized in some other dressing until the swelling subsides and the cast can then be reapplied. A mistake frequently made is to apply tight bandages over infected wounds and phlegmons. The inflammatory swelling may lead to circulatory disturbances and necrosis in a short time.

Gangrene of the fingers often develops in patients who have applied a constrictor about the upper arm, thinking that it would prevent the extension of the red streaks occurring in lymphangitis. Constriction of the penis, such as is frequently seen when a rubber constrictor or ring is applied to produce an erection, is as dangerous as paraphimosis, in which a retracted, narrow foreskin causes the circulatory disturbance. As a rule, there develops within twenty-four hours a marked venous stasis which may end in necrosis unless relieved. The necrosis may extend deeply, involving all the tissues distal to the constricting band.

Gangrene of the arm of the newborn may develop after a difficult labor in which a prolapsed arm has been pressed upon for a long time by the head incarcerated in the pelvic inlet (Fritz Müller).

Gangrene Following Strangulation, Invagination, and Volvulus.—Strangulation of intestinal loops in a hernial ring may lead to marked circulatory disturbances in a short time. The intestinal wall is then no longer able to resist the bacteria upon its mucous membrane and becomes gangrenous. The changes are most marked and develop earliest at the point of constriction.

Fæcal phlegmons developing from the hernial sac, or putrid peritonitis are the results of strangulation, unless it is relieved by early operation with resection of the gangrenous loops and end-to-end or lateral anastomosis of the resected ends.

Torsion or volvulus, by which is understood a rotation of the gut upon its mesenteric axis, interferes with the passage of intestinal contents, and, after a while, with circulation, ending in gangrene. Volvulus occurs most frequently in the sigmoid flexure, cæcum, and small intestines. Volvulus of the stomach occurs occasionally. In a similar way a piece of omentum or a pedunculated tumor may be deprived of nutrition by torsion. This occurs most frequently in ovarian tumors and pedunculated fibromyomas of the uterus. Even the uterus, when it contains fibroids of unequal size, may undergo torsion; likewise the

testicle, when it has an abnormally long mesorchium, the homologue of the mesentery, which permits of a wide range of motion, may undergo torsion and become gangrenous.

The symptoms, when the torsion is sudden, are acute and severe. The symptoms of volvulus of the intestine are those of ileus, consisting of pain, vomiting, meteorism, and obstipation. An operation should be performed as soon as the diagnosis of volvulus is made; in doubtful cases an exploratory laparotomy is advisable. If the circulatory disturbances are not so far advanced as to cause gangrene, the loops may be returned to their original position.

If the integrity of the intestinal wall has suffered, the gangrenous loop should be resected and an end-to-end or a lateral anastomosis should be made or an artificial anus established, depending upon the local and general conditions.

CHAPTER III

NECROSIS THE RESULT OF THERMAL AND CHEMICAL CAUSES

High and low temperatures (145° F. and —30° F.) may destroy even resistant tissues in a short time; lesser degrees of heat and cold destroy tissues after acting for a longer time. If gangrene develops some time after the action of heat or cold, it is due to the thrombus formation induced by them (*vide* "Burns and Frost-bites," Part IV, Chapter III).

The harmful action of Roentgen and radium rays should be mentioned here. Chronic ulcers follow the necrosis of areas of skin which have been exposed too long or improperly to these rays. The granulation tissue develops slowly, and a long time is required for healing. These ulcers are especially chronic and resistant to treatment. This is probably due to the changes in the vessel wall (endarteritis obliterans) induced by the rays (*vide* Mühsam).

There are a number of different chemical agents which may cause necrosis.

All agents used as caustics, especially acids and alkalies, destroy tissues by extracting water from or forming chemical union with the cytoplasm or interfering with circulation. The chemical agents used in sterilization may be dangerous even when diluted, especially when the protecting epithelium is absent or rapidly destroyed, when there is inflammatory hyperæmia with threatened stasis, or when the resistance of the tissues is reduced by general anæmia or weakness of the patient.

Carbolic Acid Necrosis.—The use of carbolic acid in Lister's dressing soon taught that this agent could cause severe local disturbances in the wound and skin which might end in gangrene, besides the general intoxication. Carbolic acid is still retained as the favorite germicidal agent by the laity, in spite of the fact that it has been removed from the armamentarium of the surgeon. Not infrequently a wound of the finger or toe is dressed by lay people with a moist carbolic acid compress, with the result that carbolic acid gangrene, which may even follow the application of a one per cent carbolic acid solution for twenty-four hours, develops.

Levai, Honsell, and Rosenberger have shown that this action is not specific for carbolic acid. The gangrene is due to disturbances in circulation, and is similar to that produced by other dilute chemical agents. After the carbolic acid has deprived the epithelial cells and the superficial tissues of their water, it passes between the shrunken cells to the blood vessels, which, after a transitory contraction, become dilated. As a result of the dilatation, the blood stream becomes slowed, a transudate is poured out into the subcutaneous tissues, and consequently the nutrition of the tissues is interfered with, and, as there is no absorption, the poison accumulates in the tissues. Thrombosis of the vessels is not, as Frankenburger believes, the cause, but the result of the necrosis. According to Rosenberger, coagulation of the blood is even delayed by carbolic acid.

Gangrene Following Lysol and Alcoholic Compresses.—Compresses of lysol and alcohol may have a similar action, but the gangrene does not develop so rapidly, and never extends so deeply as that produced by carbolic acid. The latter, if applied for an acute inflammatory process, such as a felon or a carbuncle, especially if evaporation is prevented by rubber tissue, may transform the entire hyperæmic area into a hard, black crust.

Destructive Action of Physiological Secretions and Excretions.—Even normal physiological secretions and excretions have a caustic action when they come in contact with exposed, unprotected, epithelial surfaces. This digestive action is occasionally seen in imperfectly performed gastrostomies from which gastric juice is discharged. An eczema then develops about the gastrostomy opening and a chronic phagedenic ulcer results from the digestion of the surrounding tissues after the protecting epithelium is destroyed. The digestive action of the gastric juice probably plays a part in the formation of ulcer of the stomach. The mucous membrane becomes necrotic as a result of thrombosis of the blood vessels or injury, and the necrotic area is then digested by the gastric juice.

The discharge of pancreatic juice into the fatty tissue of the omen-

tum, mesentery, and posterior abdominal wall following inflammation or injury of the pancreas is followed by necrosis of small circumscribed areas. After the fatty tissue is decomposed into glycerin and fatty acids, calcium salts unite with the latter to form the yellowish white nodules, the size of a pea, which are so characteristic of fat necrosis.

Urine, when it infiltrates the tissues, causes extensive necrosis. If there is an accompanying bacterial infection, a putrid phlegmon usually develops (p. 300).

Fæces contain bacteria and toxins, and severe infections with gangrene follow the escape of intestinal contents into the tissues.

Even in a bacterial inflammation the toxins are principally responsible for the death of the tissues. The injured or dead tissues in rapidly extending inflammations become necrotic or gangrenous. When the clinical course is prolonged the tissues are liquefied by the action of the leucocytes (pus formation). Interference with the circulation in these cases also contributes to gangrene of tissues, as all the vessels in the inflamed area finally become closed by thrombi.

CHAPTER IV

GANGRENE DUE TO EMBOLISM AND THROMBOSIS

Embolism and thrombosis are closely related as the causes of gangrene (*vide* Part V, Chapter IV).

Causes of Embolism and Thrombosis—Vessels Most Frequently Involved.—Emboli most frequently lodge at the point of division of an artery; for example, in the popliteal artery or in the aorta at the point of bifurcation or where the lumen of the artery is suddenly decreased by the giving off of a large branch. The emboli are carried from some part of the arterial system nearer the heart or from the left heart; only in exceptional cases in which the foramen ovale remains patent, from the right heart. Sclerosis and syphilis of the arteries, purulent arteritis in local and general infections, aneurysms and contusions of the vessel wall are the most common causes of thrombosis. Particles of a thrombus or an entire thrombus may be loosened and carried in the circulating blood to lodge in some artery more distant from the heart. Endocarditis, the result of some infectious disease, of which typhoid fever, smallpox, scarlet fever, and general pyogenic infections are the most important, is the frequent source of emboli.

Of the arteries of the systemic circulation, those of the extremities and intestines are the ones most frequently closed by emboli with sub-

sequent necrosis or gangrene. Embolism of the intestinal arteries, the symptoms of which are those of ileus, sometimes ends fatally from hæmorrhage due to infarction of the intestinal wall, sometimes from gangrene of the intestinal wall with perforation and subsequent peritonitis. Frequently embolism of these vessels is mistaken for internal strangulation or volvulus.

Symptoms.—The symptoms of embolism or thrombosis of the arteries of the extremities, those of the leg (popliteal, femoral, iliac) being much more frequently involved than those of the arm, are very distinct and definite. The symptoms begin suddenly with severe and persisting pain in the extremity involved, the skin of which becomes cold and pale or mottled. Motor and sensory paralysis develops within a few hours, just as in the gangrene which follows ligation of the principal artery of an extremity. The severest pain, however, is referred to the part which is becoming gangrenous. No pulse can be felt in the artery below the point of occlusion. The point at which the embolus is lodged is hard and painful upon pressure.

Clinical Course of Embolism.—The clinical course of embolism differs from now on. If the occlusion is complete the fate of the limb depends upon whether or not a collateral circulation is established. Frequently cardiac weakness, general arterial disease, arterial thrombosis extending from the point of lodgment of the embolus toward the heart, severe inflammation of the vessel wall caused by bacteria in the embolus prevent the rapid development of a collateral circulation which is sufficient to maintain the nutrition of the part involved. In favorable cases an œdema which persists for some time develops, the skin becomes reddened, as the smaller veins and capillaries become distended, and the symptoms of paralysis subside.

If a collateral circulation is not established, as frequently happens after traumatism of arteries, the distal parts of the extremity become gangrenous and the gangrene extends in the next few days to the level at which the line of demarcation develops.

If the occlusion is not complete from the beginning, but gradually becomes so as blood coagulum is deposited upon an embolus, the clinical picture differs. The *vis a tergo* is diminished and the small amount of blood reaching the part stagnates. The skin becomes bluish red in color, gangrenous blebs form, and ecchymoses and œdema develop.

Mummification is limited, as a rule, to the fingers and toes. The soft tissues around the demarcation zone often become gangrenous, notwithstanding that they are protected by aseptic dressings. Putrid thrombophlebitis and general infections may develop from such an area.

In rare cases, gangrene of both extremities may be of embolic origin. The embolus in these cases lodges at the bifurcation of the aorta into

the iliac vessels, and is increased by later deposits of fibrin, eventually closing both branches, or the embolus divides and pieces are carried into more distal arteries, such as the popliteal. Goedecke describes a case of embolic gangrene of the four extremities, eventually terminating fatally, which followed suppurative peritonitis.

Prognosis.—The prognosis of embolic gangrene of the extremities is always very grave. It is largely dependent upon the diseases or arterial changes which result in the formation of the embolus, and the fact that other emboli may be set loose and cause necrosis of important viscera.

Arterial Thrombosis.—Arterial thrombosis is the cause of gangrene of the extremities, when clots arising within an aneurysm developing at the site of an injury of the intima, at the point of pressure of a tumor, or resulting from acute or chronic inflammation of the arterial wall, occlude one of the larger arteries. Acute arteritis follows most frequently typhoid fever, more rarely general pyogenic infections and other infectious diseases, and is caused by the deposition of bacteria in the vasa vasorum. Cardiac weakness is also an important accessory etiological factor in the formation of arterial thrombi (marantic thrombi).

Symptoms.—The symptoms of gangrene following thrombosis of an artery are not so acute and severe as those following embolism. The symptoms develop slowly and the conditions are favorable for the development of a collateral circulation unless the anastomosing branches are also diseased. There is always more blood in the extremity than is the case when the ischæmia is due to embolism.

The dangers depend for the most part upon the disease which results in the formation of a thrombus.

Clinically it is impossible, anatomically very difficult, to determine whether the artery has been closed by a thrombus or partially occluded by an embolus upon which has been deposited masses of fibrin resulting in secondary thrombosis and complete occlusion. Relatively rapid development of gangrene speaks against chronic disease of the blood vessels.

Treatment.—The same rules should be observed in the treatment of gangrene due to embolism and thrombosis as have been given for the treatment of gangrene following ligation of larger arteries. Amputation should be postponed until the line of demarcation is well established and distinct, unless there are indications for immediate amputation, such as putrefactive changes, or unless the disease to which the embolism or thrombosis is secondary pursues a severe course. If an amputation has been made through necrotic tissue, reamputation may be performed when the line of demarcation becomes distinct.

CHAPTER V

NECROSIS RESULTING FROM CHRONIC DISEASE OF THE VESSEL WALL

Dilatation or narrowing of arteries and veins (the results of pathological processes) may lead to marked circulatory disturbances which may end in gangrene.

Aneurysms are only rarely the cause of gangrene, unless associated with thrombosis or embolism of some of the larger arteries, even when the impaired nutrition of the tissues below the aneurysm is indicated by nervous changes and the tendency to eczema. Mummification of the fingers is a frequent accompaniment of a racemose hæmangioma of the forearm.

The most frequent cause of gangrene is the narrowing of a number of the branches of an artery or of one of the larger arterial trunks by a proliferation of the intima, which, by favoring thrombus formation, may lead to complete occlusion of the vessel or vessels. These changes are most common in arteriosclerosis and syphilitic endarteritis. The arterial changes occurring in diabetic gangrene are closely related to the former.

Arterio- or angiosclerotic gangrene is called senile gangrene when it occurs in the aged; presenile, when it occurs in the young or middle-aged.

This form of gangrene occurs most frequently in men. The feet are most frequently affected, but it also develops in the hand and attacks the toes and fingers. In the beginning it attacks one foot or hand; frequently the disease develops in the other after a time. It develops gradually after prodromata, consisting of sensations of cold, numbness, neuralgic and rheumatic pains aggravated by walking when the lower extremities are involved, and of cyanosis of the feet and hands, have persisted for a long time. The frequent occurrence of this form of gangrene in the lower extremity is explained by the unfavorable circulatory conditions and the exposure of this part to mechanical insults.

Often the beginning of gangrene is indicated by the spontaneous development of a bluish black area at the tip of a toe or finger; often it follows some external violence, such as an insignificant injury, the trimming of a corn, the pressure of a boot or shoe, freezing, and not infrequently the application of a carbolic acid compress. Sometimes a dry, at other times a moist, gangrene which is frequently accompanied by lymphangitis and phlegmonous inflammations develops. The process may remain limited to the toe or finger primarily involved or extend upward, involving the larger parts of the extremities. All the fingers

or toes of an extremity may become gangrenous. When the mummified parts drop off the stumps heal.

(*a*) **Angiosclerotic and Senile Gangrene.**—Cardiac weakness is often associated with pathological changes in the walls of the arteries as an etiological factor in senile gangrene. The veins are also frequently diseased, and for this reason the term *angiosclerosis* is employed. The extent of the calcification of the arteries and the involvement of even the finer branches may be seen in Roentgen-ray pictures when the process is far advanced (Fig. 207). Cardiac weakness and arteriosclerosis are the

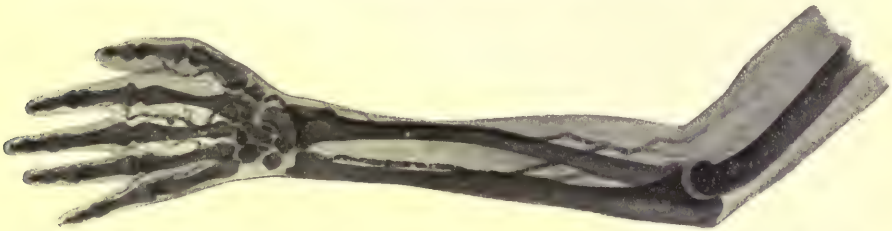


FIG. 207.—ROENTGEN-RAY PICTURE SHOWING ARTERIES OF THE FOREARM AND HAND IN ADVANCED ARTERIOSCLEROSIS. Male patient fifty-nine years of age. Dry gangrene of the terminal phalanx of the index finger.

usual predisposing causes; the determining causes may be very different. As a result of weakness or injury, a thrombus develops in the capillaries or smaller vessels or an occluding thrombus develops upon an atheromatous ulcer in one of the larger arteries. The collateral circulation is not sufficient to provide for the nutrition of the part, as the vessels entering into it are already diseased or are occluded by the growth of the thrombus.

In the presenile gangrene occurring in young people, cardiac weakness is also usually an etiological factor. Obliteration of the lumina of the vessels is due partly to the proliferation of the intima (endarteritis obliterans) and partly to the development of thrombi. Thrombi may prevent in a number of ways the establishment of a collateral circulation: (1) A thrombus may completely occlude a small diseased artery; (2) a thrombus may develop, in limited or extensive arteriosclerosis, at a point at which one of the large lateral branches is given off and occlude it; (3) multiple thrombi may develop or a thrombus may spread toward the heart, occluding even the aorta and all the branches given off from it (Weisz, Zoege, von Manteuffel, Bunge, Mantanowitsch). Excessive use of tobacco and alcohol and exposure to cold (Zoege, von Manteuffel) are important etiological factors in the development of arteriosclerosis. Rudnitzki has shown that endarteritis, with proliferation, follows repeated freezing of the extremities of ani-

mals. According to Wulff and Higier, presenile gangrene may be due to abnormal vasoconstriction similar to that occurring in Raynaud's disease.

Diagnosis.—The diagnosis of angiosclerotic gangrene should not be based upon the degeneration of arteries alone. If it is not probable that the gangrene is due to traumatism, then thrombosis or embolism, diabetes, syphilis, and nervous diseases should be excluded.

Treatment.—In the treatment an attempt should be made to prevent infection of the gangrenous area by using dry aseptic dressings. If inflammation develops, the epidermis raised by the secretion should be removed in order to prevent its extension. Moist compresses of aluminum acetate, without rubber protective, hasten the separation of the gangrenous tissue. Alcohol and carbolic acid compresses should never be used, as these agents alone may cause gangrene.

Phlegmons require incision. If a phlegmon does not subside after incision and if the condition of the patient becomes rapidly worse, immediate amputation is indicated. Great care should be exercised in the application of a constrictor for artificial ischæmia. It should be applied where the artery is well protected by muscles, and in all cases it is well to apply a heavy towel or a bandage about the extremity before applying the constrictor, thus taking extra precaution against injury.

If the arteriosclerosis is advanced, it is well to perform the amputation without a constrictor, using digital compression of the artery or catching the vessels with artery forceps as they are exposed or cut. The amputation should be performed rapidly and, if used, the constrictor should not be allowed to remain any longer than is absolutely necessary. If allowed to remain any length of time it may cause an extension of the gangrene. Skin flaps and bone flaps should not be made because of the dangers of necrosis. If the patient has myocarditis or nephritis, a general anæsthetic should not be used; local or spinal anæsthesia should be employed instead.

(*b*) **Diabetic gangrene** is closely related to angiosclerotic gangrene. [" It is mainly due to the abnormal condition of the blood in diabetes, thereby reducing the power of the tissues to resist bacterial invasion, but is also, in a measure, the result of sclerosing endarteritis and peripheral neuritis."—Rose and Carless, " Manual of Surgery," p. 113.] The resistance of the tissues in diabetes is so reduced that pyogenic and putrefactive infections extend rapidly and cause rapid and extensive destruction of tissue, even when the vessels are not diseased. Infections extend much more rapidly and are much more destructive when, in addition to the lowered resistance of the tissues, there are circulatory and nutritional disturbances, the result of pathological changes in the walls of the arteries.

Lowered Resistance of the Tissues.—The following example of the rapid progress and fatal termination of an infection, which ordinarily could easily have been controlled, may be cited. A student twenty years of age, suffering from diabetes, was shot in the back while hunting. An acute, rapidly extending gangrene of the skin and muscles, which soon terminated fatally, rapidly developed, although there were no pathological changes in the arteries supplying the part.

Infections in Experimental Diabetes.—The animal experiments made by Bujwid, Groszmann, and Hildebrandt, concerning the relation between diabetes and infections are interesting and instructive. They have shown that micro-organisms multiply much more rapidly in and are more pathogenic for diabetic than non-diabetic animals. A subcutaneous injection of a streptococcic culture into animals suffering from experimental diabetes produces a severe inflammation associated with gangrene of the tissues and ulcer formation, while the same amount injected into a normal animal is absorbed and produces no symptoms.

The facts that inflamed tissues in diabetic subjects rapidly become gangrenous, that suppuration is rare, and that when it does occur but little pus is formed indicate that the resistance of the tissues to bacterial infections is greatly reduced (*vide* Pyogenic Infections).

Fig. 208.—Diabetic Gangrene.

Clinical Course of Diabetic Gangrene.—Clinically the relation between gangrene and inflammation is much more striking in diabetic than any other variety of gangrene. Diabetic gangrene may begin, as an arteriosclerotic gangrene usually does, in the skin of one of the toes. Prodromata, which have already been described in discussing senile gangrene, may have been present for some time. Inflammation then develops in the gangrenous area, and, as a result, uncomplicated dry gangrene or mummification is rare in this disease. The reverse may happen; that is, inflammation develops first, often at some point of pressure, in a fissured callous (upon the heel, sole of the foot, or in the palm of the hand) or after excision of a corn, and gangrene develops secondary to the inflammation.

As a result, diabetic necrosis or gangrene is rarely limited, and

separation of the dead tissues with spontaneous healing rarely ever occurs. Diabetic spreads much more rapidly than senile gangrene, as infection is frequent and it may destroy a large part of the foot or leg.

Complications.—The dangers of diabetic gangrene are those associated with the rapid extension of the gangrene and the development of phlegmons which may cause general infections. Complications, which may develop at any time in the clinical course of diabetes, add to the gravity of the prognosis. The most important of these will be briefly mentioned: (1) Attacks of acute cardiac weakness, caused as a rule by arteriosclerosis, which, after a short febrile reaction, may end in fatal collapse. (2) Diabetic coma, beginning with prodromata consisting of nausea, restlessness, and headache, followed shortly by severe nervous disturbances (stupor, delirium, and unconsciousness) and ending fatally in a few hours or days. [Fatal coma may develop when general anæsthesia is administered to diabetic patients. In this connection it is interesting to note that acetone and beta-oxybutyric acid are found in the urine of a majority of patients to whom chloroform is administered. Ether seems to have less effect upon metabolic processes than chloroform; beta-oxybutyric acid is not found so frequently after this anæsthetic. Becker, especially, has drawn attention to the dangers of administering chloroform to diabetics.] Psychical irritation may follow operations upon diabetic subjects even when performed under local anæsthesia.

Treatment: Prophylactic and Surgical.—In the treatment of diabetic gangrene an attempt should be made to check the elimination of sugar by prescribing a suitable diet and by internal medication. The diet suggested by von Noorden is very valuable in these cases. The indications for surgical interference depend entirely upon the course of the gangrene and the general condition of the patient. The extremity should always be immobilized in well-fitting and padded splints and elevated in order to combat the lymphangitis (*vide* General Rules for Treatment of Infections, p. 196) and improve the circulation.

Dry gangrene, which is rare in diabetes, should be treated according to the rule already given for the treatment of senile gangrene. Moist compresses, however, should never be used in diabetic gangrene, as they favor the development of putrefactive processes (König). If putrefaction occurs, hydrogen peroxid should be used.

Incision of a gangrenous phlegmon is sufficient if more radical measures are not indicated by the general condition of the patient; for as the glycohæmia subsides under anti-diabetic treatment, and as the general condition of the patient improves, the inflammation subsides and the gangrene becomes limited. Dry dressings favor mummification, moist dressings, with the exception of those of hydrogen peroxid, do

harm. The epidermis raised by collections of pus should always be removed. If, however, the phlegmonous inflammation spreads and the gangrene extends in spite of incision, if a high fever persists, if the amount of sugar is not decreased, if the general condition of the patient becomes worse, or if from the beginning there is great weakness, symptoms of cardiac insufficiency, or prodromata of diabetic coma, amputation is indicated. Recovery occurs in about sixty per cent of the cases (Groszmann). The point at which the amputation should be performed depends altogether upon the extent of the inflammation and the disease of the arterial wall. If the phlegmon has extended to the leg, or if the popliteal artery is occluded and transformed into a hard cord, a thigh amputation should be performed. If the gangrene is of the dry variety and limited, a Pirogoff or Chopart's amputation or an exarticulation of the gangrenous toes may be sufficient.

If gangrene develops in the flaps after an amputation and extends, reamputation at a higher level must be performed.

The operation should be as simple as possible (circular amputation without formation of flaps is best) and should be performed under the strictest aseptic precautions. Touching of the wound surfaces with the fingers should be particularly avoided. The Esmarch constrictor, if the arteries are sclerotic, should be allowed to remain in position as short a time as possible. Many surgeons use merely digital compression of the principal arteries when performing amputations under these conditions. On the other hand, it should be mentioned that the loss of even a small amount of blood may prove dangerous when the arteries are diseased and the heart is weak. If there is lymphangitis, the wound should not be sutured, but should be tamponed with iodoform gauze.

Dangers of General Anæsthesia in Diabetic Patients.—A general anæsthetic should not be administered to a diabetic patient unless it is absolutely necessary, and should not be continued any longer than is required for the performance of the operation. Frequently a general anæsthetic (especially chloroform) increases the acetonuria and glycosuria. Not infrequently diabetic coma follows general anæsthesia. According to Becker, chloroform and ether, especially the former, cause in healthy men a slight and transient acetonuria (Kausch).

Even local anæsthesia by infiltration is not without danger in these cases. The infiltrated tissues may become gangrenous, and besides the operation cannot be performed so expeditiously and there is greater danger of infection. Frequently the patients become nervous and greatly excited, and this may lead to fatal syncope or diabetic coma (as has happened in von Bergmann's clinic). Spinal anæsthesia is well suited for these cases, as it is borne well and permits of rapid, uninterrupted amputations of the lower extremities. [Nitrous oxid gas

is the anæsthetic of choice in these cases.] Subsidence of the fever and decrease in the amount of sugar after the amputation are always favorable signs. Treatment is practically useless after coma has developed.

Acid Intoxication.—It has been suggested that diabetic coma is caused by an intoxication with acids, especially beta-oxybutyric acid, the result of abnormal metabolic processes (Stadelmann). When this intoxication occurs, the blood is no longer able to form a chemical union with the carbon dioxid in the tissues and to remove it. Acting upon this theory, attempts have been made for a number of years to prevent the accumulation of these abnormal acids in the blood by administering alkalies, especially sodium bicarbonate by mouth or in a three to five per cent solution intravenously. Kausch has recently recommended the administration of sodium bicarbonate before operations upon diabetic patients, or when coma is threatened or has developed. He administers large doses, as much as 150 gm. in twenty-four hours.

(*c*) **Syphilitic gangrene** of the extremities is the result of syphilitic changes in the larger arteries and their branches, with secondary thrombosis. Syphilitic endarteritis occurs both in congenital and acquired syphilis.

It is difficult to estimate the frequency of this form of gangrene, and it is frequently confused with gangrene of the extremities due to other causes, especially with the presenile form of angiosclerotic gangrene.

It occurs more frequently in men than in women and attacks the young and middle-aged. Clinically it does not differ from the arteriosclerotic gangrene occurring in young people. The prodromata are the same and of as long duration. The gangrene, which is as a rule limited in extent, develops upon the fingers and toes, frequently in a number of different areas, and may occasionally be symmetrical (Elsenberg). It may be associated with phlegmonous inflammation. After the necrotic tissue is cast off, chronic ulcers which resist treatment form. The large arteries of the extremities are transformed into hard, pulseless cords in which may be demonstrated circumscribed nodular thickenings.

According to Haga, who with Scriba has emphasized the frequent occurrence of syphilitic disease of the larger arteries in the Japanese, besides the obliterating growth of the intima, foci of granulation tissue develop in the walls of the artery which correspond to gummata. These changes have also been described by Baumgarten in the cerebral arteries.

Syphilitic disease of the arteries should always be thought of when gangrene of the fingers and toes develops in young people (under thirty-five years) slowly, after years of circulatory disturbances. This is especially so when there are indications of a congenital or an acquired syphilis. A definite diagnosis can be made only when the circulatory

disturbances and the other symptoms subside under anti-syphilitic treatment, or a microscopic examination of the arteries of the amputated limb can be made.

The same rules concerning dressings and indications for operation as have been given for angiosclerotic gangrene should be followed. Of course, anti-syphilitic treatment with mercury preparations and potassium iodid should be tried before more radical measures are instituted.

(*d*) **Varicose Veins.**—Circulatory disturbances, the result of dilatation of the veins, are of very frequent occurrence in the leg (*vide* Phlebectases).

Predisposing and Determining Causes of Varicose Veins.—Impairment of the venous circulation alone, due to insufficiency of the valves of the dilated veins, is not sufficient to cause necrosis of the skin. In the changes associated with varicose veins, however, the regenerating power of the tissues, especially those covering the anterior surface of the tibia and the malleoli, are so reduced that any lesion, such as a contusion, a scratch, an abrasion, or an infection, does not heal rapidly, but tends to spread and form an ulcer. Impairment of venous circulation is a predisposing cause in the development of varicose ulcers; there are a number of determining causes, such as abrasions, contusions of the cutaneous and subcutaneous tissues, infiltration of the tissues with blood (the result of the bursting of a varix), infection of the skin, secondary to suppuration of a thrombus, and hyperæmia associated with a furuncle by which the venous circulation is still more impaired. Wherever any of these lesions occur, there may be a superficial and limited necrosis of the skin, which extends, resulting in the development of the typical varicose ulcers.

The development and extension of these ulcers, the difficulty in securing permanent repair, and the frequent recurrence after healing are due to causes other than the impairment of circulation following dilatation of the veins. Some of these causes will be mentioned: (1) Impairment of circulation from excessive walking or too long standing, which are required in so many callings and professions. The small veins, already filled with blood, rupture and small hæmorrhages occur in the floor of the ulcer or into the delicate scar which is already forming, resulting in the necrosis of tissue and extension of the ulcer. (2) All varieties of bacteria are deposited upon the surface of the ulcer. Infection occurs, the ulcer becomes larger and deeper, and gangrene, which causes a rapid extension of the necrotic process, may develop. The necrosis is due not only to the bacterial toxins, but also to the inflammatory hyperæmia of the surrounding tissue, which, as the circulation of the tissues is already impaired, leads to rapid stasis in and thrombosis of the inflamed veins. Acute progressive inflammations, such as

erysipelas, lymphangitis, and thrombophlebitis, may result in occlusion of the veins by thrombi and interfere with the return flow of blood. (3) In old ulcers large amounts of cicatricial tissue form, which may even extend into the tissues surrounding the ulcer.

The thin skin becomes adherent to the underlying structures and the edges of the ulcer become indurated and fixed. If the ulcer extends completely around the leg, the contraction of the cicatricial tissue may interfere still more with the venous circulation, causing a marked chronic œdema, with elephantiasis of the skin of the foot. [Occasionally the interference with venous circulation is so great that moist gangrene develops, and an amputation must be performed.]

Most Common Location of Varicose Ulcers.—Varicose ulcers develop most frequently upon the internal and anterior surfaces of the lower third of the leg and about the internal malleolus; that is, in the area drained by the long saphenous vein. More rarely they develop in the area drained by the short saphenous vein.

Frequently varicose ulcers are bilateral. A varicose ulcer may be present upon one leg, and on the other an eczema, covered with scales and crusts, which terminates later in ulceration, or a dark-brown pigmented scar resulting from the healing of a previous ulcer.

Small ulcers may develop in any patient afflicted with varicose veins. They rarely develop, however, before the fortieth year. Large ulcers are seen more frequently in poor people. Hard work and uncleanliness are important etiological factors.

Appearance of Varicose Ulcers.—Varicose ulcers differ in appearance, but they are characteristic enough to enable one to differentiate between them and syphilitic or carcinomatous ulcers. The edges of the ulcer are irregular, not undermined, slope gradually to the floor of the ulcer or are indurated and hard. The ulcer is shallow, unless its edges are swollen and œdematous, and then the floor of the ulcer is deep. The floor of the ulcer is covered with a yellow, caseous *débris* or granulation tissue which is only in rare instances healthy. The granulations even in old ulcers are exuberant, flabby, cyanotic, or dotted with small

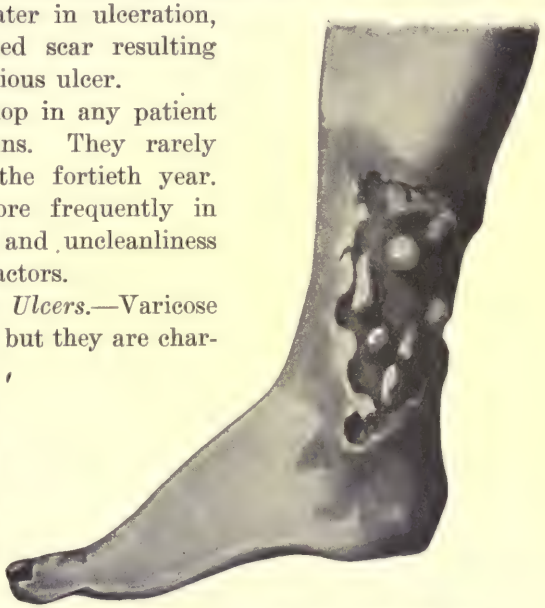


Fig. 209.—Varicose Ulcer of the Leg.

hæmorrhages. The secretion from the ulcer is profuse, being seropurulent or putrid in character. Adjacent to the ulcer may be found a delicate, brown, pigmented scar, with shining or scaling surface, which is adherent to the deeper tissues, or a squamous or pustular eczema. The surrounding skin is, as a rule, œdematous or hypertrophied. Inflammatory redness is rarely absent.

Differential Diagnosis.—Syphilitic ulcers, due to regressive changes in cutaneous or periosteal gummata, are so characteristic that they are easily distinguished from varicose ulcers. It is not always so easy to make a differential diagnosis between a varicose and carcinomatous ulcer, as occasionally a carcinoma develops upon an old varicose ulcer. The rapid development of nodular, hard granulations in a varicose ulcer should always arouse suspicion. The edges of varicose ulcers may become raised by the development of masses of cicatricial tissue and resemble those of a carcinomatous ulcer.

Fig. 210.—Syphilitic Ulcer of the Leg.

The growth of a varicose ulcer is rarely continuous and progressive. Healing and ulceration alternate in the clinical picture. The ulcer, which in the beginning is about as large as a ten-cent piece and scarcely extends to the corium, may heal if favorable conditions are provided. The epithelial islands still remaining between the papillæ proliferate to cover the ulcer. A delicate, brown, pigmented scar develops, the circulation and nutrition of which are poor. This scar breaks down from almost any cause (e. g., exertion, inflammation, and trauma) and an ulcer larger and deeper than the former develops. Alternating changes, repair and ulceration may go on for a number of years. The development of large amounts of cicatricial tissue favors ulceration and the extension of the pathological process. Finally the ulcer may extend half way or completely around the leg, and become as wide as the hand.

The pain in varicose ulcers may be very severe. Ulcers situated about the internal malleolus give rise to the severest pain, which is aggravated by walking. The pain is often so severe that the patient is compelled to hold the foot in supination while walking. Old ulcers may give rise to functional disturbances as the cicatricial tissue extends deeply and involves the tendons and ligaments. This process may result in anchylosis of the ankle joint. The foot then is not much better as regards function than an artificial foot (Nasse).

Varicose ulcers resemble very closely ulcers which develop upon the leg as the result of impairment of circulation due to excessive walking and long-continued standing. These ulcers are not associated with varicosities. Frequently thrombophlebitis and lymphangitis develop from such ulcers; the veins become dilated as a result, and in this way ulcer formation is favored.

Treatment.—The following rules may be given for the treatment of varicose ulcers: (1) Improve the circulation of the extremity by rest and elevation. If the patient is up and about, advise the use of a bandage which supplies gentle elastic compression. Flannel and jersey bandages, bandages and stocking of elastic webbing are to be preferred. The latter, however, prevent evaporation and favor the development of eczema. The large dilated vein should be resected or the main trunk of the long saphenous vein ligated or resected at the saphenous opening, providing its radicals are involved. [Before operative treatment is instituted a careful history should be elicited regarding the probable etiological factors. If the deep veins are occluded or dilated, the operation should not be performed, as the entire venous circulation will then be thrown upon the deep veins, the blood will stagnate, and moist gangrene of the extremity will develop. The deep veins are always involved when the lesion occurs after typhoid fever and phlegmasia alba dolens; the femoral vein is involved in the former, the iliac veins in the latter.]

(2) Bacterial infections should be prevented by cleanliness, frequent application of aseptic dressings, and sterilization of the area surrounding the ulcer. The ulcer should be protected from external irritation (e. g., rubbing of clothes) by copious, properly applied dressings. Moist, antiseptic dressings of aluminum acetate favor the development of granulation tissue, the separation of gangrenous tissue, and the removal of irritating bacterial products. Cauterization of the ulcer with silver nitrate, removal of flabby granulations with the sharp spoon, and finally excision of the indurated cicatricial borders favor repair.

Old chronic ulcers should be excised and the fresh defect should then be covered with skin grafts. Cutis grafts are used more extensively than epidermal grafts for this purpose. The extremity still becomes congested when it is dependent or used in walking, and epidermal grafts do not do well, even when precautions are taken to prevent passive hyperæmia of the limb during the process of repair and for some time afterwards. Pedunculated flaps taken from the area adjacent to the ulcer or from the other leg heal more readily than non-pedunculated flaps.

Unna's zinc-gelatin dressing, as used by Heidenhain, is recommended for patients who must be up and about tending to business.

The dressing exerts even compression upon the skin, takes up the secretion of the ulcer and protects it. After a warm footbath and sterilization of the skin of the leg, eczematous areas are covered with Lassar's paste, ulcers with iodoform gauze, or, if granulation tissue has already developed, with salve. A zinc-gelatin mixture (zinc oxid, gelatin, āā 20.0; glycerin, distilled water, āā 80.0) is melted over a water bath and then the foot and leg are painted with it. A gauze bandage is then applied smoothly and evenly and another coating of the zinc-gelatin mixture is then applied. Four layers of bandage and four coatings of gelatin mixture are applied alternately, and then an ordinary mull bandage is applied. The dressing, which hardens in twenty-four hours, should be removed as soon as the secretion of the ulcer soaks through it.

Sometimes when the ulcers are large and chronic, especially when they occur in old people, amputation must be considered. Amputation is indicated when protracted putrefaction leads to cachexia and amyloid degeneration of viscera is feared, when general infection develops or a carcinoma develops upon an ulcer.

CHAPTER VI

NEUROPATHIC GANGRENE

Ulcers of the skin and mucous membranes, mummification or gangrene of the terminal parts of the extremities may follow diseases or injuries of the central nervous system or of the peripheral nerves. Nutritional disturbances, due to the loss of trophic influences, certainly play a part in this form of gangrene, but other influences, such as transitory or continued pressure, injuries, and inflammation cannot be excluded.

Tissues deprived of their nerve supply become necrotic more frequently than normal tissues, and the necrosis develops more rapidly and is more extensive. This is probably due to a number of factors: (1) the resistance of the tissues to bacterial infections is reduced; (2) the circulation is impaired as a result of vasomotor disturbances; (3) the area supplied by the injured or diseased nerve is, as a rule, anæsthetic, and as injuries and inflammations are no longer painful the lesions resulting from them are neglected.

Neuroparalytic Keratitis.—In many cases *keratitis neuroparalytica*, accompanied by ulcer formation, follows inflammation and paralysis of the trigeminal nerve (resulting from injury, tumors, aneurysms, resection of the Gasserian ganglion for trifacial neuralgia). The anæsthetic

cornea is more exposed to external influences (dust, contact with the finger) than the normal, and its epithelium is easily injured. Its resistance to infection is reduced and a simple conjunctivitis may be the cause of a severe keratitis, resulting disastrously. Ulceration of the anæsthetic mucous membrane of the tongue and cheek is usually the result of injuries by the teeth.

Sometimes ulcers, at other times dry gangrene, develop upon the trunk and extremities as the result of diseases and injuries of the central and peripheral nervous system. The disease or injury is the predisposing cause, but usually there are a number of determining causes. The necrosis, however, is never extensive, unless moist gangrene develops.

Acute Decubitus in Paraplegia and Hemiplegia.—Acute decubitus develops frequently in hemiplegias and paraplegias of whatever origin (cerebral and spinal tumors, hæmorrhages into the spinal cord, or injuries).

It may develop upon any part exposed to slight pressure, most frequently in the sacral region, the back, or over the heel. The lesions may become quite extensive, as the area is anæsthetic and they are not discovered until late unless the doctor or nurse anticipates them. In locomotor ataxia and paralysis of the lower extremities, especially in spina bifida, dry, painless ulcers (perforating

Fig. 211.—Decubitus in a Paralytic Club Foot Associated with a Spina Bifida Occulta with Hypertrichiasis.

ulcers), which resist treatment, develop in the sole of the foot. The ulcers develop from a callosity, a fissure, or an insignificant injury. Similar ulcers, which, however, are usually accompanied by severe pain, may develop in neuritis. In syringo-myelia and the nervous form of leprosy, painless, dry gangrene as well as ulcers due to pressure necrosis develops upon the fingers. In leprosy the toes may also be involved. After the separation of the necrotic parts the involved extremity is crippled and transformed into a short cicatricial stump. After injury of the nerves of the extremities, small, deep, painless ulcers may develop upon the palmar surfaces of the fingers and the sole of the foot from ruptured vesicles or blebs.

Raynaud's Disease.—Symmetrical gangrene, or Raynaud's disease, and perforating ulcer of the foot (*malum perforans*, Nélaton) are peculiar forms of gangrene and ulcer, the etiology and exact classification of which are not at all clear. It is doubtful whether it is correct to maintain that they are distinct clinical entities.

The form of gangrene designated as Raynaud's disease develops in young (especially anæmic) people, also in children. In this disease a dry, usually symmetrical, gangrene develops upon the fingers, more rarely upon the toes, the ears, the cheeks, and the nose. It begins with nervous and vasomotor disturbances, similar to those to be described later in ergotism.

Pallor and coldness of the part involved, the symptoms of local ischæmia (*local syncope*), follow the prodromal stage of severe pain and paræsthesias (formication and numbness). The pallor and coldness may disappear or the skin may become bluish red in color (*local cyanosis, asphyxia*), and necrosis, usually of the terminal phalanges only, with vesicle and crust formation, develops. The entire course of the disease may extend over months and years and be combined with scleroderma. Roentgen-ray pictures indicate the disappearance of the terminal phalanges. Nothing definite is known concerning the vascular spasm which is peculiar to the beginning of Raynaud's disease. Inflammatory changes have been found in the vessels and nerves, but they are not constant. According to Cassirer, the irritability of the vasomotor tracts and centers is increased as the result of some congenital predisposition or some frequently repeated harmful influence, such as cold, infectious diseases, or poisoning—for example, with chloral and phosphorus. Severe psychical disturbances (fright) have also been suggested as etiological factors. Usually there is no disease of the central nervous system. Symptoms of Raynaud's disease may develop, however, during the clinical course of locomotor ataxia, syringo-myelia, epilepsy, hysteria, exophthalmic goiter, and tumors of the spinal cord (Oppenheim).

Sometimes when there is but little pain and the symptoms of the

disease are not pronounced, it is very difficult to differentiate Raynaud's disease from syringo-myelia and leprosy. Both syphilitic and angiosclerotic gangrene may be symmetrical, and as they may be accompanied by severe pain, they may resemble closely gangrene occurring in Raynaud's disease.

Ergot Gangrene.—Gangrene occurring in chronic ergot poisoning (ergotism) is probably due to angiospasm. Cases of ergot gangrene are rarely seen at the present time (except in Southern Russia, Zoege von Manteuffel), although it was very common in the Middle Ages. It follows the use of diseased rye in the making of bread. The severe cases end fatally in a few days, the symptoms consisting of sensory disturbances (formication, pain, or anæsthesia), convulsions, vomiting, and diarrhœa. In the milder cases the symptoms of ischæmia develop especially in the distal parts of the lower extremities, as a result of the contraction of the walls of the blood vessels. Pain is first noted in the parts involved which later become pale, cold, and anæsthetic. As the disease advances the parts become cyanotic, and then a dry or a moist gangrene develops. Billroth describes a case of ergot gangrene of the fingers which followed the administration of large doses of this drug for medicinal purposes.

Perforating Ulcer.—By perforating ulcer (*malum perforans pedis*) is understood a chronic, painless form of ulcer which resists treatment and recurs without any apparent cause. It may develop upon any part of the foot, most commonly, however, at the three points on the plantar surface which are constantly exposed to pressure when the patient stands or walks. These three points are the heads of the first and fifth metatarsal bones and the heel. The area surrounding the ulcer is anæsthetic or analgesic, and there are, as a rule, trophic changes in the skin and nails. Necrosis follows a suppurative inflammation which develops beneath a callosity, in a small wound or an accessory bursa. The necrosis progresses slowly and a deep, funnel-shaped ulcer with precipitous and undermined edges forms. It may heal temporarily and then break down again. Repair and ulceration may alternate for a number of years, and the ulcer may eventually afford the infection atrium for a severe putrefactive

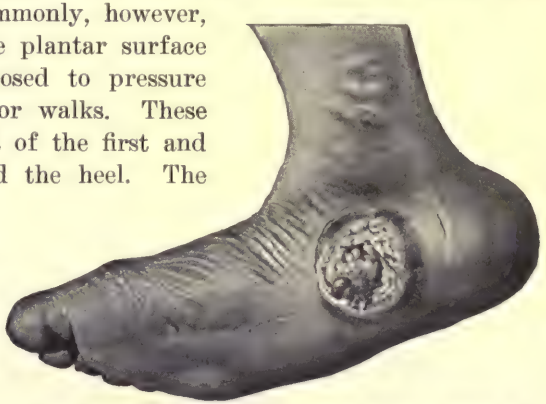


Fig. 212.—Perforating Ulcer of the Foot in Locomotor Ataxia.

infection ending in disorganization of neighboring joints and destruction of bone.

Perforating ulcers follow diseases and injuries of the central nervous system and peripheral nerves (for this reason they are called neuroparalytic ulcers by Duplay, Morat, H. Fischer, and others). They are frequently found in locomotor ataxia, syringo-myelia, spina bifida (Fig. 211), injuries and diseases of the vertebræ with involvement of the spinal cord, paretic dementia, injuries, tumors, and inflammation of the nerves. Disease of the vessels, which is sometimes found, cannot be regarded as the cause of the ulcer (Nasse).

This form of ulcer also occurs in diabetes as a result of an accompanying neuritis. It differs from diabetic gangrene proper in the absence of pain.

Ulcers due to other causes—for example, old, neglected wounds and ulcers occurring in angiosclerosis and syphilis—may be easily differentiated from perforating ulcers, as they are painful and there are no accompanying sensory disturbances.

The small multiple foci of cutaneous necrosis (multiple neurotic necrosis of the skin, herpes zoster gangrænosus, Kaposi, gangrenous urticaria, Renaut), which may be distributed over the entire surface of the body are probably due to changes in the nervous system. Hysterical patients occasionally produce a necrosis of the skin with caustic soda, which resembles this form very closely (Cassirer).

Treatment.—The first indications in the treatment of necrosis of neuropathic origin are to remove the cause when possible and to treat the principal disease. Otherwise the same rules that have been given for the treatment of angiosclerotic gangrene should be followed. The cicatricial edges of the ulcers should be excised in order to provide good drainage for the secretion; moist dressings and hot baths favor the development of granulation tissue and healing. Necrotic bone should be removed, joints destroyed by suppuration, resected. Recurrence may follow excision of the ulcer. According to Chipault, permanent healing may be secured if at the time the ulcer is excised the nerves about the ankle are stretched.

Literature.—*Adrian.* Das Mal perforant. Centralbl. f. Grenzgeb., 1904, p. 321.—*Barraud.* Ueber Extremitätengangrän im jugendlichen Alter nach Infektionskrankheiten. Deutsche Zeitschrift f. Chir., Bd. 74, 1904, p. 237.—*Beck.* Some New Points in Regard to Raynaud's Disease. Amer. Journal, November, 1901.—*Bergmann* (*Morian*). Ueber Gefässverletzungen in der Kniekehle. Festschr. zur Huyssenstiftung, Essen, 1904.—*v. Bergmann.* Die isolierte Unterbindung der Vena femoralis communis. Würzburger Festschr., 1882;—Die Schussverletzungen der Art. subclavia, etc. St. Petersburger Wochenschr., 1877.—*Brandweiler.* Multiple neurotische Hautgangrän. Monatshefte f. prakt. Dermatol., Bd. 39, No. 5.—*Cassirer.* Ueber multiple neurotische Hautgangrän. Zentralbl. f. Grenzgeb., 1900, p. 161.—*Chipault.* Radikalbehandlung

des Mal perforant durch Nervendehnung. Gaz. des hôp., No. 41, 1901.—*Elsenberg.* Die sogenannte Raynaudsche Krankheit (Grangraena symmetrica) syphilitischen Ursprungs. Arch. f. Dermatol. und Syphil., Bd. 24, 1892, p. 577.—*Erb.* Ueber Bedeutung und praktischen Wert der Fussarterien bei gewissen anscheinend nervösen Erkrankungen. Mitteil. aus d. Grenzgeb., Bd. 4, 1900.—*Fraenkel.* Ueber Verletzung der Vena fem. communis und deren Behandlung. Beitr. z. klin. Chir., Bd. 30, 1901, p. 81.—*Frankenburger.* Ueber Karbolgangrän. I.-D. Erlangen, 1891.—*Franz.* Zur Unterbindung der Vena femoralis am Lig. Poup. Deutsche militärärztl. Zeitschr., 1903, Hft. 9.—*Goedecke.* Spontane Gangrän an den vier Extremitäten. Verhandl. d. freien Chirurgenvereinig. Berlin, 1903, Jahrg. 16.—*Groszmann.* Ueber Gangrän bei Diabetes mellitus. Berlin, 1900, Hirschwald.—*Haga.* Ueber spontane Gangrän. Virchow's Arch., Bd. 152, 1898, p. 26.—*Higier.* Zur Myasthenia paroxysmalis angiosclerotica und der sog. spontanen Gangrän. Zentralbl. f. Chir., 1901, p. 1080.—*Hildebrandt.* Ueber diabetische Extremitätengangrän. Deutsche Zeitschr. f. Chir., Bd. 72, 1904, p. 351.—*Honsell.* Ueber Karbolgangrän. Beitr. zur klin. Chir., Bd. 19, 1897, p. 623.—*Höpfner.* Ueber Gefässnaht, u. s. w. Arch. f. klin. Chir., Bd. 70, 1903, p. 417.—*Kausch.* Beiträge zum Diabetes in der Chirurgie. Chir.-Kongr. Verhandl. 1904, II, p. 655.—*Lévai.* Ueber Mal perforant du pied. Deutsche Zeitschr. f. Chir., Bd. 49, 1898, p. 558.—*Matanowitsch.* Zur Kasuistik der Spontangangrän. Beitr. z. klin. Chir., Bd. 29, p. 545, 1901.—*v. Mikulicz* und *Kausch.* Embolie und Thrombose der Mesenterialgefässe. Handb. der prakt. Chir., 2. Aufl., Bd. 3, p. 386.—*Mühsam.* Doppelseitige Oberschenkelamputation wegen akuter Gangrän. Deutsche Zeitschr. f. Chir., Bd. 70, 1903, p. 333.—*Fritz Müller.* Ueber Gangrän von Extremitäten bei Neugeborenen. I.-D. Strassburg, 1900.—*Nasse.* Krankheiten der unteren Extremitäten. Deutsche Chir., 1897.—*Oppenheim.* Lehrbuch der Nervenkrankheiten. Berlin, Karger.—*Raynaud.* De l'asphyxie locale et de la gangrène symétrique des extrémités. Paris, 1862.—*Rotter.* Die Stichverletzungen der Schlüsselbeingefässe. v. Volkmann's Samml. klin. Vortr., N. F., No. 72, 1893.—*v. Recklinghausen.* Handb. der allgem. Patholog. des Kreislaufes und der Ernährung. Deutsche Chir., 1883.—*Roosen-Runge.* Ueber die Bedeutung des Traumas in der Aetiologie der disseminierten Fettgewebsnekrose. Zeitschr. f. klin. Med., Bd. 45, p. 418.—*Franz Rosenberger.* Ursachen der Karbolgangrän (experimentelle Untersuchungen). Würzburg, Stuber, 1901.—*v. Wartburg.* Ueber Spontangangrän der Extremitäten. Beitr. z. klin. Chir., Bd. 35, 1902, p. 624;—Ueber das Mal perforant des Fusses. Ibid., Bd. 36, 1902, p. 212.—*O. Weber.* Von dem Brande. v. Pitha und Billroth's Handb. der Chir., 1865, 1. Bd., 1. part.—*H. Wolf.* Diabetische Gangrän und ihre Behandlung. Sammelreferat. Zentralbl. f. Grenzgeb., 1901, p. 21.—*Wormser.* Ueber puerperale Gangrän der Extremitäten. Wien. klin. Rundschau, 1904, Nos. 5–6.—*Wulff.* Ueber Spontangangrän jugendlicher Individuen. Deutsche Zeitschr. f. Chir., Bd. 58, 1901, p. 478.—*Ziegler.* Ueber Stichverletzungen der grossen Gefässe der Extremitäten. Münch. med. Wochenschr., 1897, p. 733.—*Zoege v. Manteuffel.* Die Arteriosklerose der unteren Extremitäten. Mitt. aus dem Grenzgeb., Bd. 10, 1902.

PART IV

INJURIES OF THE SOFT TISSUE, BONES AND JOINTS AND THEIR TREATMENT

An injury is an alteration in the tissues induced by the application of some force, the results of which are the separation of the injured tissues from their normal connections, an alteration of their normal structure, or a disturbance of nervous activity such as is seen in shock. The injurious agents (the so-called traumas), as well as the results following their action, are widely different. Injuries are classified as mechanical, chemical, and thermal.

I. THE MECHANICAL INJURIES

CHAPTER I

GENERAL CLASSIFICATION OF MECHANICAL INJURIES

We recognize the following forms of injuries: contusions, lacerations, and concussions, and special varieties occurring in certain systems only, such as fractures of bones, dislocations of joints, and subluxations of tendons and nerves.

A contusion is an injury which follows a compression of the tissues beyond their powers of resistance. The action of blunt force or the striking of a part of the body upon a hard object are the most usual causes. The soft parts are either compressed against the bone, or are caught between two resistant surfaces—the most usual mechanism when the larger parts of the body are injured—which are forced together like the arms of a pair of tongs. The contusion is limited to those tissues acted upon by the compressing surfaces or to all those compressed between the object and projections or edges of the bone. The skin, being more resistant than fat and muscle, may remain practically intact, even when the subjacent soft tissues are transformed into a pulpy mass and are separated from all their normal connections.

A **contusion** is not only a compression of the tissues, but is also, and according to Gussenbauer, is principally, a laceration of the tissues which follows a stretching beyond their greatest point of elasticity.

According to Gussenbauer, in the mildest contusions the changes are limited to a separation of the loose connective tissues and a tearing of the smallest lymphatic and blood vessels, while in the severer contusions the intercellular substance is lacerated. He finds that the greater part of the cellular elements may be retained even in the severest crushing injuries, in which the tissues may be completely separated from each other.

The *common symptoms* of a contusion are pain, loss of function, and swelling which follows hæmorrhage from the torn vessels. Associated with these symptoms is the discoloration of the surface following changes in the extravasated blood.

Lacerations are injuries which follow stretching of the tissues by a wrench or traction, the blunt force being either applied obliquely or acting from within the body. If the tissues are stretched beyond their own degree of elasticity, the intercellular substance, rarely the parenchyma proper, is lacerated. If the gross anatomical structure is preserved, one speaks of a stretching or tearing; if the tissues are separated, of a rupture. If the skin is torn, the wound is spoken of as a lacerated wound, and when the force has acted from within, as a bursting wound.

Pain, impairment of function, and swelling due to the extravasated blood are the most important symptoms.

Concussion is the term employed to indicate the condition induced by the action of blunt force (thrust, blow, fall) which is not powerful enough to cause demonstrable changes in the tissues, such as a solution of continuity. The effects of the concussion may extend immediately to the large parts of the body or remain limited to the part to which the force was applied, in which case it is usually associated with a contusion or laceration. The symptoms, consisting of pain, partial or complete loss of sensation and motion, are due to alterations in nervous tissue. Concussion of the tissues adjacent to contused wounds is common, the symptoms being grouped under the term *local shock* or *wound stupor*. The symptoms associated with a concussion soon disappear unless the tissues are contused or lacerated.

Examples of concussion are known to all through personal experience. When one jumps some distance and alights upon a hard surface, the lower extremities being held stiff, a concussion is experienced. A dull pain passes through the legs, which are momentarily numb and palsied. The numbness quickly gives way to a tingling sensation, and the latter, after a time, to a feeling of weakness. Similar sensations may be experienced following a slight injury of the ulnar nerve behind the internal

condyle, the sensory disturbance extending even to the area supplied by the terminal filaments of the nerve.

Concussion of the brain and the rarer forms of concussion of the spinal cord, thorax, and abdomen are more dangerous, and not infrequently end fatally. In these cases the symptoms are rarely due to the concussion alone, but are combined with those due to severe crushing injuries and internal hæmorrhage, and may pass imperceptibly into those of severe inflammation.

The chief *symptoms* of concussion of the brain following injuries of the skull are loss of consciousness and defects of memory following the return of consciousness. These are due to some injury of the nerve centers, the exact nature of which is unknown. There is a good deal of doubt concerning concussion of the spinal cord, and the cases which have been regarded as such were probably contusions or lacerations of the cord following hyperflexion of the spinal column. Reflexes play an important part in concussions of the thorax and abdomen, and for this reason they are frequently classified with shock. In these cases there is a paralysis of the vasomotor apparatus or a stimulation of the cardio-inhibitory centers caused by irritation of the vago-sympathetic, splanchnic, or sensory nerves.

Frequently an injury cannot be placed in any of the groups above mentioned, as it may be a combination of two or more varieties. For example, a fragment of a bomb may contuse and lacerate the tissues and produce a concussion at the same time. A bullet may injure the soft tissue and pass to the bone. The latter is comminuted, and the soft tissues are not only injured by the bullet, but by the fragments and fractured ends of the bone as well; concussion, laceration, and contusion of the tissues being combined. A laceration is always associated with a contusion when the force is applied obliquely to the surface of the body.

LITERATURE.—*Gussenbauer.* Die traumatischen Verletzungen. Deutsche Chir., 1880.

CHAPTER II

MECHANICAL INJURIES OF THE DIFFERENT TISSUES

I. INJURIES OF THE SKIN, SUBCUTANEOUS TISSUES AND MUCOUS MEMBRANES

BESIDES the simple wounds, which have already been described (Chapter I), **excoriation** and **contusion of** and **extravasation of blood into** the skin also occur. Excoriations are most usually produced by a fall,

being observed frequently upon the knees and chins of children, or are associated with injuries produced by blunt force. The epidermis is scraped away when the part glides upon the floor after a fall or when blunt force is applied tangentially. The corium, which bleeds easily and is painful, is then exposed, being frequently covered by gross particles of dirt. The exposed surface soon becomes covered with a crust as the blood and tissue fluids rapidly coagulate. Healing without scar formation occurs beneath the crust, providing suppurative or phlegmonous inflammation, lymphangitis, and erysipelas do not develop.

In the *treatment* the grosser particles of dirt (sand, splinters of wood) which have been forced into the tissues should be removed with tissue forceps. The excoriation should then be washed with a three per cent solution of hydrogen peroxid, after the adjacent area has been carefully sterilized. A dry aseptic dressing should be applied to prevent infection if the crust falls off, and especially to protect against injury by scratching and removal of the crust when the injured area itches.

Simple contusions of the skin pursue different clinical courses. When the skin lies directly over a bone, no soft tissues intervening, the injury may be severe enough to render the area bloodless and insensitive, necrosis developing later. On the other hand, a contusion of the skin may be followed by a subcutaneous hæmorrhage, indicated by the appearance of a discoloration which disappears slowly, and is due to the extravasation of blood into the loose tissues or by the development of a bleb with hæmorrhagic or serohæmorrhagic contents (blood blister). In order to prevent infection, these blebs should not be opened, but should be protected with dry aseptic gauze attached by adhesive strips. They dry up after a few days. A new epithelium develops beneath the old epidermis and the crust, which gradually become separated. Injured blebs should be treated like those developing in gangrene—i. e., they should be removed and the resulting wound should then be covered with a dry aseptic dressing.

Petechiæ, Suggillations, Hæmatomas.—The subcutaneous tissues may be contused and torn as the result of the action of blunt force without any injury of the elastic and easily displaceable overlying skin. An extravasation of blood, which either infiltrates or separates the loose tissues, is the result of such an injury. A hæmorrhagic infiltration follows bleeding from capillaries and numerous small vessels; a hæmatoma (blood tumor), bleeding from a single artery or vein of small or large diameter. If the hæmorrhagic foci are small and form more or less sharply defined, punctate, red or reddish black spots, we designate them as petechiæ or ecchymoses; if they are larger and less clearly defined, as suggillations and as bloody effusions. If the skin is closely related to bone, the fascia and periosteum, which are usually also injured, are

infiltrated with blood. In hæmophilia very insignificant injuries may be followed by the development of large hæmatomas.

The hæmorrhage from the injured vessels usually subsides rapidly, for the lumina are closed by the pressure and the coagulation of the extravasated blood. Coagulation is never complete in large hæmatomas, as the extravasated blood is mixed with lymph, which coagulates very slowly and interferes with the coagulation of blood.

Absorption of a Blood Clot.—An hæmorrhagic infiltration is absorbed more rapidly and completely than a hæmatoma. The serum which is separated during coagulation of the blood is absorbed first, a large number of leucocytes being carried with it into the adjacent lymph nodes. The clot itself is gradually prepared for absorption by the digestive action of ferments liberated by the leucocytes and the cells of the surrounding tissues. Hæmoglobin liberated by the degeneration of red blood cells penetrates with the serum the surrounding tissues, causing discoloration. The greater part of the hæmoglobin which is set free is finally absorbed and excreted by the liver as bile pigments and through the kidneys. Some of it, after having undergone a modification, remains in the tissues as a yellow, granular iron-containing pigment known as *hæmosiderin* and as *hæmatoidin* crystals, which are ruby red in color and needlelike or rhomboid in shape. These derivatives of hæmoglobin may be found within the cells or lie free in the tissues.

While these changes are going on, the fibrin and injured and dead tissues are being absorbed, infiltrated, and replaced by granulation tissue. The resulting connective-tissue mass—the *scar*—is usually firmer than the surrounding tissues.

Symptoms of a Subcutaneous Hæmorrhage.—The symptoms of a subcutaneous hæmorrhage are moderate pain, a bluish red discoloration of the skin, which is more marked the looser the tissues (being most marked, for example, in the eyelids and scrotum), and the development of a more or less circumscribed swelling which fluctuates or crepitates, depending upon whether the blood remains fluid or coagulates. Pulsating hæmatomas develop after the injury of large arteries (*vide* p. 552). Circumscribed hæmatomas are characterized by the gradual induration of their edges, due to the formation of a clot. An aseptic fever may be associated with the larger extravasations of blood.

Clinical Course.—The pain and swelling disappear after several days or weeks, depending upon the amount of blood which has been poured out into the tissues. The discoloration may extend over a wide area within the first week; for example, the discoloration associated with a hæmatoma beneath the skin of the shoulder may extend over the entire upper arm and adjacent side of the chest, increasing in size as the hæmoglobin penetrates the various layers of tissue. The discoloration

is at first of a purplish hue; later it becomes bright green and finally yellow, the latter color being the most persistent.

Treatment.—The application of a bandage exerting mild compression is the best treatment that can be employed during the first few days, provided there is no inflammatory reaction. It relieves pain, promotes absorption, and prevents further hæmorrhage which might possibly occur during movements. The ice bag and cold applications are to be especially recommended in the treatment of subcutaneous and deep hæmorrhages. Massage is of value when the hæmorrhage has ceased, as it hastens the absorption of the clot. Aspiration is indicated if the hæmatoma is absorbed slowly; incision, if suppuration occurs.

Blood Cysts.—Larger hæmatomas in the subcutaneous as well as in the muscular and retroperitoneal tissues, likewise small extravasations in the brain, frequently pursue a clinical course different from that described above. Absorption does not take place rapidly from the connective-tissue wall lined with fibrin surrounding the blood clot, and the fluid part of it becomes transformed into a brownish red, chocolate colored, thin or thick mass which contains blood pigment, hæmatoidin and cholesterin crystals, the last being apparently derived from degenerating fat. The wall of the cavity eventually becomes transformed into thick fibrous tissue which may later become calcified, or, if situated near a bone, ossified. Traumatic blood cysts are formed in this way. If aspiration of the contents and subsequent injection of some irritating substance, such as alcohol, iodin, carbolic acid, or iodoform emulsion, do not cause the obliteration of such a cyst, it should be incised and its walls partially or completely removed.

Post-operative Hæmatomas.—A hæmatoma may develop in an operation-wound, following hæmorrhage from some vessel which has not been ligated. The fluid blood then trickles from the spaces between the stitches and saturates the dressings; the clots separate the tissues forming cavities, and cause severe pain by exerting pressure upon them. If a hæmatoma develops in an operation-wound, the skin surrounding it should be sterilized, a few or all of the sutures removed, and the clots expressed by digital pressure applied to the edges of the wound. During the manipulation the fingers should not come in contact with the wound. If the bleeding vessel is found, it should be ligated. The wound should then be tamponed with iodoform gauze and no attempt should be made to suture it for several days. Carelessness in the control of hæmorrhage is frequently followed by the development of a hæmatoma. Severe inflammations frequently develop in such wounds, as infection may be easily introduced during the removal of the clot.

In all operative work blood vessels should be grasped with artery forceps as soon as cut, for the walls of the smaller vessels collapse, and

they are then found with more or less difficulty. These arteries are often opened again when the patient awakes and the blood pressure becomes higher. Careful hæmostasis prevents the development of hæmatomas. Post-operative hæmatomas develop quite frequently after infiltration anæsthesia, as the cutaneous veins are closed by the pressure of the solution and are consequently overlooked.

Subcutaneous Separation of the Skin.—The severest injuries of the subcutaneous tissues are those associated with a separation of the skin from the subjacent tissues. The skin may be torn loose from the subjacent fascia by force acting obliquely (in railroad and machine injuries). Blood and lymph are then poured out beneath the loosened skin, elevating the latter to form a tense, fluctuating swelling. Laceration of the muscles, blood vessels, and nerves, and injuries of the bone may occur at the same time. A large hæmatoma or an extravasation of lymph forms in these cases, and some of the characteristic signs—swelling, discoloration of the skin, fluctuation, and crepitation—are rarely absent.

The characteristic physical findings associated with an extensive subcutaneous separation of the skin were first described by Morell-Lavallée under the term of *décollement de la peau*, and later by Koehler. The findings differ from those associated with the typical extravasations of blood described above. The swelling develops gradually, often several days intervening before it reaches its maximum size, while the swelling associated with a typical hæmatoma develops rapidly. The fluid poured out beneath the separated skin is usually serum, although at times there may be a small amount of blood. As the lacerated lymphatic vessels are not closed by thrombi, the extravasation continues until the pressure of the exuded lymph equals the intravascular lymphatic pressure. As the latter is low the swelling never presents the resistance associated with hæmatomas, but imparts a flaccid, relaxed sensation. The shape of the swelling varies with changes in the position of the body, and a distinct wave of fluctuation can be elicited by tapping the swelling with the finger. The separation of the skin associated with an accumulation of lymph occurs most frequently upon the thigh; occasionally upon the trunk (Fiebiger). Besides this separation of the skin (superficial décollement), there also occurs a stripping of the muscles and periosteum from the bone (deep décollement).

The treatment of subcutaneous separation of the skin begins with careful sterilization of the area involved and the aseptic dressing of all excoriations. A compression bandage, when properly applied, favors absorption. The skin, however, is deprived of a number of its nutrient arteries, and necrosis is apt to occur unless the dressing is applied evenly and changed often. The larger accumulations should be removed

by aspiration before the dressing is applied. Accumulations of lymph may be injected with alcohol or a five per cent solution of iodin before the dressing is applied. Incision is to be recommended only when necrosis or inflammation is beginning. Phlegmons find favorable conditions for development in these cases. Frequently they pursue a severe clinical course after contusions.

A complete separation of the scalp from the subjacent tissues has been reported (*vide* Altermatt). This accident occurs most frequently in people who work about machines, the hair being caught in the belts connecting the driving wheels. The entire scalp may be torn off and the resulting defect must then be skin-grafted. Separation of the skin covering the penis and scrotum has also been observed in accidents caused by machinery.

Injuries of Mucous Membranes.—Injuries of the mucous membranes are quite similar to those of the skin, except that wounds and excoriations of the former are not followed by such virulent infections as are those of the latter. However, a submucous hæmorrhage following a gunshot wound or a subcutaneous injury may, if the larynx is involved, cause a laryngeal stenosis and threaten life. If the stomach or intestines are involved a fatal perforative peritonitis may develop, as a result of the nutritional disturbances, ending in necrosis and perforation, caused by the pressure of the blood clot.

Traumatic Emphysema.—Traumatic emphysema presents a rather characteristic clinical picture. It develops after injuries of the air passages, when the expired air is forced into the meshes of the loose subcutaneous tissues and into the cellular tissues surrounding muscles, vessels, and viscera.

The mild forms, in which the swelling is not so marked, are associated with injuries of the nose, frontal and maxillary sinuses, and mastoid process. It develops in injuries of the nose when the patient attempts to remove blood clots by blowing. The severer cases follow perforation of the lung by a fractured rib, bayonet, or projectile, injuries and inflammatory (tuberculous) perforation of the larynx and trachea, especially when the wound canal is narrow and oblique.

An emphysema may develop after tracheotomy if the air escapes by too narrow an outlet. I have seen a slight subcutaneous emphysema develop after a laparotomy performed in the Trendelenburg position, the air in the abdominal cavity being forced by vomiting or coughing into the subcutaneous tissues on either side of the wound. Frequently emphysema develops after wounds of the soft tissues, the air having been forced into the tissues through a drain or during irrigation. An emphysema may develop in wounds produced by blank cartridges, the

shot being fired at very close range and the gases formed during the explosion being driven into the tissues (Hammer, Schaefer).

A subcutaneous emphysema is characterized by development of a soft, elastic swelling with indistinct boundaries which has a tympanitic note, crackles when palpated, and is painless. This swelling may increase rapidly in size, especially when the patient is restless, and spread over large areas. In marked cases the emphysema may involve the entire skin, which then becomes inflated like a balloon and transformed into a tense, tympanitic, crackling mass.

The localized forms of emphysema are the most common. In these cases the emphysema is limited to a small area, the swelling attains its greatest size in a few days and gradually subsides as the air is absorbed.

The greatest danger associated with the most marked forms of emphysema is extension to the mediastinum. For example, a case has been observed in which the infiltrating air extended from a wound of the thorax over the entire trunk, neck, face, and extremities, finally beneath the pleuræ, and from the loose connective tissues of the neck into the mediastinum. The latter was already affected as a result of an injury of the trachea and larynx before the subcutaneous emphysema developed. The respiratory and cardiac functions were interfered with and death followed, the symptoms being those of suffocation.

Traumatic emphysema is readily differentiated from gas phlegmon, as in the former all the symptoms of local and general infection are wanting.

The *treatment* consists of closing the wound from which the air is escaping, when possible, or of incising the tissue and permitting of the escape of air, thus preventing the infiltration of more tissue. The nose may be closed with tampons. Wounds of the larynx and trachea should be found and closed by sutures. Penetrating wounds of the chest associated with pneumothorax should be tamponed, or, if this is not successful, the opening should be enlarged and the opening in the parietal pleura closed. If the latter is not successful or the air is discharged from a lung which is adherent to the chest wall, the wounds in the soft tissues should be dilated and a drainage tube, provided with a valve which permits of the escape of air during expiration (von Bramann), inserted (König). The same procedures should be followed in treating emphysema developing after injuries of the lungs caused by fractured ribs, when compression does not suffice to prevent the emphysema. It is often possible to prevent the spreading of an emphysema by making free incisions into the tissues primarily involved (König).

Literature.—*Altermatt.* Ein Fall von totaler Skalpierung. Beitr. z. klin. Chir., Bd. 18, p. 765, 1897.—*v. Bramann.* Ueber die Bekämpfung des nach Lungenverletzung auftretenden allgemeinen Körperemphysems. Chir. Kongr.-Verhandl., 1893. Disk.

König.—*Cordua.* Ueber den Resorptionsmechanismus von Blutergüssen. Preisschrift. Berlin, 1877.—*Fiebiger.* Ein Fall von subkutaner traumatischer Lymphorrhagie. Wien. klin. Wochenschr., 1897, No. 17.—*Gussenbauer.* Die traumatischen Verletzungen. Deutsche Chir., 1880.—*Hammer.* Traumatisches Hautemphysem durch Pulvergase. Beitr. z. klin. Chir., Bd. 25, 1899.—*Jutkowski.* Ueber plastische Operationen an Penis und Skrotum im Anschluss an einen Fall (Kausch) von Schindung der männlichen Genitalien. I.-D. Breslau, 1904.—*Klaussner.* Studie über das allgemeine traumatische Emphysem. München, 1886.—*Köhler.* Ueber Morell-Lavallées Décollement traumatique de la peau et des couches sousjacentes. Deutsche Zeitschr. f. Chir., Bd. 29, 1889.—*Madlena.* Das Hautemphysem nach Laparotomien. Monatsschr. f. Geb. u. Gynäk., Bd. 13, 1902.—*Raviant* et *Marlier.* Emphysème souscutane de la face, du cou et du tronc. Gazette hebdomad., 1899, No. 38.—*Schäfer.* Ueber Hautemphysem nach Schussverletzung. Beitr. z. klin. Chir., Bd. 28, 1900.

II. INJURIES OF FASCIA AND MUSCLES

(a) SUBCUTANEOUS INJURIES

A subcutaneous laceration of fascia may be caused by the sudden contraction of the muscle or muscles which it covers. The fascia of the biceps brachii is ruptured most frequently when an attempt is made to prevent a heavy load from falling. The fascia of the adductors is ruptured quite frequently during horseback riding, the rider attempting to maintain himself in position by contracting the adductor muscles when the horse suddenly shies or kicks. Usually the fascia is lacerated over the belly of the muscle, but occasionally it gives way at other points, especially if the fascia has been previously injured by a fractured bone.

Muscle-Hernia.—The rent in the fascia may be felt, when the muscle is at rest, as an irregular, oblique defect in which a circumscribed soft swelling (which increases in size and becomes hard when the muscle contracts) can be palpated. This so-called muscle-hernia causes some distress at first, but later occasions but little or no pain, and interferes but little with the function of the muscle.

Operative closure of the rent in the fascia is therefore but rarely indicated. If indicated, the tear may be closed by passing sutures through the edges of the fascia, including also some of the muscle fibers, thus securing a firm grasp of the tissues. At times, in repairing large muscle-herniæ, it may be necessary to remove the projecting part of the muscle, preliminary to closing the fascia. Small rents in the fascia demand no treatment.

A muscle-hernia is often simulated by the bulging of a circumscribed thin area in the fascia when the muscle contracts.

Fascial injuries due to contusions are often associated with similar injuries of the muscles.

Contusion of Muscles.—Muscular contusions are caused by severe falls and blows. They are especially apt to occur when muscles are pressed

against bones. They may also be caused by the bites of animals (horse-bites). A slight contusion is merely followed by an infiltration of blood into the muscle involved. In the severer injuries part of the muscle is destroyed, considerable blood is poured out into the wound, and a brownish red, pulpified mass (muscle hæmatoma) is formed.

The injured muscle then no longer contracts normally, and when attempts at contraction are made fibrillary twitchings occur. Disturbed function, severe pain, and swelling, the size of which depends upon the amount of hæmorrhage, are the most important symptoms of a contusion of a muscle. Fluctuation can be distinctly elicited only in the large hæmatomas. There is no discoloration of the skin, unless the fascia and subcutaneous tissues are also injured.

The prognosis varies. Frequently all the symptoms disappear in a few weeks. The destroyed muscle tissue is replaced by granulation tissue, which is soon transformed into a firm scar (myositis traumatica fibrosa). This scar tissue remains permanently, notwithstanding the fact that after a time a few regenerating muscle fibers infiltrate it. It does not, however, interfere with the function of the muscle, unless a large part of the latter has been destroyed. If this has happened a contracture which may seriously interfere with the function of the part develops. Ossification of a muscle (myositis ossificans traumatica), which may follow a single or repeated trauma, and infection either through the lymphatics or blood are rare sequelæ. Extensive destruction of a muscle is more frequently followed by the formation of large encapsulated hæmatomas or extravasations of lymph, the tissues surrounding which become calcified or ossified, than by scar formation. These do not interfere greatly with function, but are troublesome because of their size and weight. The symptoms and prognosis of a contusion involving the entire thickness of a muscle are the same as those of a laceration.

The early treatment consists of massage and moderate compression obtained by a properly applied bandage. Massage should be employed later, the pain and swelling frequently disappearing and motion returning within two weeks after it is begun. Traumatic blood and lymph cysts require repeated aspiration. In some cases a radical operation—complete removal of the cyst—is required. Contractures may be corrected by tenotomy and function improved by uniting detached ends of muscles to adjacent healthy tendons or muscles, when this procedure is indicated and possible.

Laceration of Muscles.—A laceration of a muscle may be complete or incomplete, and affect the belly of the muscle or the point of transition of muscle fibers into the tendon. Lacerations may be produced in a number of ways. They may follow excessive stretching of a resting

muscle; for example, laceration of the sterno-cleido-mastoid muscle in difficult breech presentations, of the adductor muscles of the thigh in forceful attempts to reduce a congenital dislocation of the hip under anæsthesia. In other cases the muscle fibers are lacerated when the muscle contracts powerfully; for example, when an attempt is made to lift a heavy weight. A muscle is much more easily lacerated in a contracting than in a resting state. The following examples which almost always occur in muscular, well-built men may be cited:—Rupture of the rectus abdominis or the quadriceps extensor in attempting to prevent a fall backward; of the quadriceps extensor or the muscles of the calf in jumping from some distance or in springing up suddenly from the floor. Very frequently the biceps brachii (most commonly the long head) is ruptured in lifting a heavy weight. The muscles about the shoulders, in the neck, back, or abdominal wall are quite often lacerated by sudden overextension or torsion of the trunk.

A muscle may also be ruptured by direct violence, but this mechanism is much rarer than that cited above. A diseased muscle (degeneration associated with and following such diseases as typhoid fever, scarlet fever, smallpox, general infections, acute miliary tuberculosis, etc.) may rupture even when it is not overstretched or forcibly contracted; for example, the rectus abdominis may rupture at the first attempts of a convalescent typhoid patient to walk. Lacerations of muscles, like contusions, are frequently associated with other injuries, such as fractures with a wide displacement of fragments and dislocations.

The symptoms of a rupture of a muscle are not always distinct. Sudden, lightninglike pain in the muscle involved and complete loss of function are the most positive symptoms. Often the patient hears at the time of the accident a distinct snap. If the rupture is complete the gap in the muscle can be distinctly palpated at first. Later a hæmatoma develops and fills this in. When attempts at movement are made both ends of the muscle contract if the innervation is still preserved. If the rupture is incomplete an indistinct swelling due to the accumulation of blood which is limited to the muscle develops.

Repair as in contusions is followed by the development of scar tissue. If the rupture is incomplete, repair may be established in one or two weeks. When the rupture is complete the ends of the muscle may become united by this scar tissue and the function completely reëstablished. If the development of the scar tissue is excessive, a hard fusiform swelling forms. This is the muscle callus which gradually contracts and becomes smaller, often producing deformities. Wryneck following injuries of the sterno-cleido-mastoid muscle at birth is a common example.

The treatment of an incomplete rupture of a muscle is the same as that of a contusion. If the rupture is complete an attempt should be

made to approximate the two ends by immobilizing the parts in the proper position. The plaster-of-Paris dressing which is applied for this purpose should be allowed to remain from four to six weeks. Suturing of the ends of the ruptured muscle is a much more certain procedure than the one just mentioned, and should always be advised when the space between the divided ends is great. It should not be delayed too long. The immobilizing dressing applied after suturing should be allowed to remain for three weeks. If muscle suture is delayed the ruptured ends retract and atrophy, and can no longer be brought together. Transplantation of muscle is rarely successful in these cases, for the tissue rapidly degenerates and is of no value in filling in the defect.

Massage and active motion are important in the after-treatment.

(b) OPEN INJURIES OF FASCIA AND MUSCLES

Depending upon the character of the vulnerating force these wounds are either incised, lacerated, or contused.

The results depend upon the amount of functional disturbance and the development of inflammation. The functional disturbances following punctured and gunshot wounds, superficial incised wounds, and contusions are slight and transitory, unless there is an injury of some motor nerve. On the other hand, the results following injuries produced by a hatchet, a saber, a bayonet or knife, the explosion of a bomb, or inflicted by some animal are severe and permanent unless proper treatment is instituted. Infection develops in a muscle wound with frayed edges very easily, while clean-cut wounds are inclined to heal by primary union. The small wound of entrance associated with punctured and gunshot wounds favor healing without infection.

The treatment usually does not differ from that of a simple wound. Fascia and muscle which have been divided should be sutured. Buried catgut or silk sutures should be employed, and the divided ends of the muscles should be accurately approximated, so that no dead spaces are left. After the muscle is sutured, the fascia should be united with some superficial sutures. The scar which follows healing by primary union is insignificant, and almost completely disappears after a short time. If the wound is not very recent it is a good plan not to close the skin completely, but to insert a tampon of iodoform gauze which extends to the line of suture, and can be replaced by a tubular drain if infection occurs. In recent contused and lacerated wounds and in compound fractures the divided muscles should be sutured after the severely injured muscle tissue has been removed. If the wound is not recent, it should be left open and allowed to heal by granulation tissue. If the functional disturbance following contraction of the scar is great the results may be improved by a plastic operation upon the muscle.

LITERATURE.—*Féré.* Les accidents de l'attaque d'épilepsie liès à la contraction musculaire. Revue de Chirurgie, T. 21, 1900, p. 50.—*Loos.* Ueber subkutane Bicepsrupturen. Beitr. z. klin. Chir., Bd. 29, 1901, p. 410.—*Marchand.* Der Prozess der Wundheilung. Deutsche Chir., 1901. Heilung der Muskelwunden, p. 289.—*Maydl.* Subkutane Muskel- und Sehnenzerreissungen. Deutsche Zeitschr. f. Chir., Bd. 17.—*Steudel.* Zur Behandlung und Operation der Muskelbrüche. Beitr. z. klin. Chir., Bd. 34, 1902, p. 611.

III. INJURIES OF TENDON

(a) SUBCUTANEOUS INJURIES

Contusions of tendons occur only when great force is applied (machine injuries, kicks by horses, etc.). Tendons are very resistant to the milder forms of trauma, and as they frequently lie upon soft tissue, they are pushed aside and escape injury when the vulnerating force acts. Crushed tendons are frayed and fibrillated. If the tendon lies in a tendon sheath, an extravasation of blood may occur in the latter, when the corresponding tendon is contused or lacerated by the contraction of the muscle to which it is attached, or by the powerful contraction of an antagonistic muscle. These injuries are frequently associated with fractures and dislocations.

Rupture of Tendons.—Rupture of a tendon occurs less frequently than the corresponding injury of a muscle. In rupture of a tendon the solution of continuity takes place more frequently at the point of insertion of the tendon into the bone and at the junction of the muscle with the tendon than in the tendon proper. If rupture occurs at the point of attachment of the tendon to the bone, a piece of the latter is usually torn off, and the tendon is frayed at the point of rupture.

Tendons are usually ruptured by the rapid contraction of powerful muscles; less frequently by direct force acting when the muscle is at rest. Trauma, direct or indirect, associated with a dislocation may be the direct etiological factor in the rupture of a tendon. Long-continued inflammation and repeated mechanical insults (von Volkmann's fragilitas tendinum) may be predisposing factors. Softening of the tendons combined with fibrillation which is associated with certain occupations (e. g., changes in the extensor longus pollicis of the left hand of drummers and the development of a ganglion) may favor rupture of the tendon or tendons involved.

Tendons Most Frequently Ruptured.—The tendons of the quadriceps extensor and of the biceps brachii are ruptured most frequently. The ligamentum patellæ may be ruptured at its point of attachment to the tibia or to the patella. Fibers of the quadriceps extensor tendon may be ruptured at their points of attachment to the patella or to the muscle. In rare cases the tendinous tissue is ruptured both above and below the patella. The long head of the biceps may be ruptured

at its middle or torn away from its point of attachment to the scapula. The tendo Achillis may be torn away from its point of attachment to the os calcis; the tendon of the triceps from its point of attachment to the olecranon process. The attachments of the extensor tendons of the fingers to the terminal phalanges are occasionally torn loose when the distal phalanges are forcibly flexed and the two proximal phalanges remain extended. The attachments of the superficial and deep flexor tendons of the fingers may be torn away with pieces of the bones by a sudden powerful contraction of the extensors when the fingers are flexed.

Symptoms.—The rupture of a tendon is frequently indicated by a sharp audible snap which is accompanied by pain. Loss of the function of the muscle involved; a change in the position of the extremity or part of the extremity (e. g., flexion of the terminal phalanx following rupture of the attachment of the extensor tendon); loss of resistance between the ends of the tendon, or between the tendon and the bone, as the case may be; and displacement of the distal end of the belly of the muscle in a direction opposite to the one which it normally passes when contraction occurs are the most important signs of a rupture of a tendon. As a rule, the hæmatoma which develops after such an injury is not very large.

If a tendon is ruptured an operation should be performed as soon as possible, and the defect in the tendon, between the tendon and bone, or between the tendon and the muscle repaired by suture. The extremity should then be placed in an immobilizing dressing to prevent contraction of the muscles. The part should be placed in a position which relaxes the muscles when the dressing is applied. Immobilization should be continued from four to six weeks. The distal end of the long head of the biceps may be sutured to the short head if the ruptured ends have retracted so much that approximation is impossible. If the ends of ruptured tendons have retracted so much that it is impossible to unite them, a tenoplasty should be performed. Frequently the scar which develops between the sutured ends of a tendon stretches, and the function of the muscle is permanently impaired, unless another operation is performed, the scar tissue is removed and the vivified ends of the tendon are sutured again.

Subluxation of Tendons.—By subluxation of a tendon is understood a change in the position of a tendon following rupture of its synovial sheath and restraining bands. Subluxation of tendons is rare. It occurs most frequently about the external malleolus, the tendon of the peroneus longus or the tendons of both peronei muscles being displaced forward after rupture of their synovial sheaths and restraining bands. The retinacula and sheaths are most frequently torn in the latter case by

sudden contraction of the peronei muscles when the foot is in the position of supination. Subluxation of the tendon of the tibialis posticus has been observed but once. Subluxation of the long head of the biceps may occur after rupture of its sheath, together with the capsule of the joint and the attachment of the supraspinatus muscle. This occurs only with fractures of the neck of the humerus or dislocations of the shoulder (König). Subluxation of the extensor tendons of the fingers to the ulnar side of the heads of the metatarsal bones, after rupture of the bands passing from the radial side of the dislocated to the adjoining tendon, has been occasionally observed (Schuermayer, Haberern, Becker). A similar subluxation of the tendons may occur in chronic articular rheumatism. The extensor tendons may then be displaced so far forward that they might act as flexors instead of extensors (Krukenberg).

Diagnosis and Treatment.—The displaced tendon may easily be felt in its abnormal position, and easily returned to the position which it should occupy. If it is possible to hold the tendon in its proper position for a number of weeks by a gauze or cotton pad over which an immobilizing dressing is applied, healing in normal position may occur. If the function of the part is interfered with and conservative treatment has been unsuccessful, an operation should be performed. If the old sheath can be found, the tendon should be replaced and the margins of the sheath sutured over it. If the sheath cannot be found a new canal should be made out of the surrounding connective tissues, the tendon placed in it, and the connective tissues sutured over it, forming a substitute for the normal restraining bands. Flaps of fascia, or, according to König, a periosteal-osteal flap when the tendons of the peronei muscles are dislocated, may be used for this purpose.

(b) OPEN INJURIES OF TENDONS

Wounds associated with partial or complete division of tendons occur most frequently on the hands. They are less common on the feet and other parts of the body. These injuries are usually produced by sharp instruments or objects, such as knives, pieces of metal and glass, or are associated with contusions and lacerations caused by machinery, the explosion of shells or boilers, and by the bites of animals. They are often associated with wounds of vessels, nerves, bones, and joints, or with more extensive wounds, the injury of the tendons then being of secondary importance. The ends of the tendons are clean cut, contused, or fibrillated, depending upon the character of the wound. The entire tendon, even up to its origin from the muscles, may be torn away in the severer injuries caused by machinery.

The divided ends cannot always be seen in the wound, even after it has been carefully cleaned, and its margins held apart with hook

retractors. They retract from the wound, the proximal end naturally retracting the most. Those tendons with long synovial sheaths and the greatest range of motion retract the most. The distal end retracts less than the proximal. Even when a flexor tendon is injured when the part is in flexion and is subsequently extended, the distal retracts less than the proximal end.

Diagnosis.—If a division of a tendon is suspected, the function of that tendon should be tested. This should always be done in injuries adjacent to a tendon, even if the wound is very small.

Treatment.—An attempt should always be made to perform a primary tendon suture in wounds of this character, unless the injury to the tendon or the character of the wound contraindicates such a procedure. Severely contused and frayed tendons, such as are frequently associated with contused and lacerated wounds, are not at all suited for primary suture as necrosis occurs, and besides the resistance of the tissues is so reduced that infection is apt to occur. It is often better in these cases to delay suturing until the conditions are more favorable for repair. It is even dangerous to search for and to suture the ends of tendons in wounds which have been improperly treated or neglected, and in which suppuration has already developed. Suppuration is followed by necrosis at the line of suture and by changes in the tendon sheaths, which may be followed, if the flexor tendons of the fingers are involved, by a progressive phlegmon extending rapidly to the fascial spaces of the forearm or by contractures.

If the wound is not clean or cannot be properly prepared, primary tendon suture should not be performed. The wound should be treated by the open method until healthy granulation tissue has developed or the wound has healed. Then a secondary tendon suture should be performed as soon as possible. In these cases it is always difficult to find the proximal ends of the tendons as they have retracted, and the space between the divided ends is often so great that it is impossible to unite them.

Incomplete division of a tendon is difficult to recognize, as there is no loss of function. The defect is usually easily recognized, however, when the wound is being treated.

Tendon Suture.—The part should be rendered bloodless before an attempt is made to find the ends of the tendons, which are more readily found in recent than in old wounds in which considerable scar tissue has already formed. The distal ends may be found by hyperflexion or hyperextension of the part, as the case may be, while the proximal ends may be made to appear by bandaging or massaging the belly of the muscle involved toward the wound. In cases in which the ends have retracted a great deal, it may be necessary and advantageous to

incise the tendon sheath in both the proximal and distal directions. The incision should be made to one side of the tendon in order to prevent union between the two layers of the synovial membrane. In old cases it may be necessary to make a rather extensive dissection in order to find the ends of the tendons. The scar tissue should be removed.

The ends of the tendons should be anchored by a silk suture as they are found. This suture should be applied so that it can be used later in uniting the divided ends. If a number of tendons have been divided, as frequently happens in cuts involving the flexor surface of the wrist, it may be difficult to identify the ends which should be united. The appearance, position and size, anatomical relations, and function of the divided ends indicated when traction is made upon them all aid in recognition of the separate ends.

The ends of crushed and lacerated tendons should be resected before they are united.

When the other end is found, the suture which has already been inserted in the end previously found should be passed through the former. The suture should then be tied, the muscle to which the tendon is attached being held in a relaxed position in order to prevent the sutures cutting out. The ends must be accurately coapted; a side-to-side approximation avoided.

Sublimate silk should be used in suturing tendons. A number of different methods of tendon suturing have been devised. The simplest are the best. I employ the method devised by Friedrich (Fig. 213) in suturing small tendons, that devised by Haegler (Fig. 214, *b*) in suturing large ones. The sutures recommended by Woelfler and Trnka are more complicated.

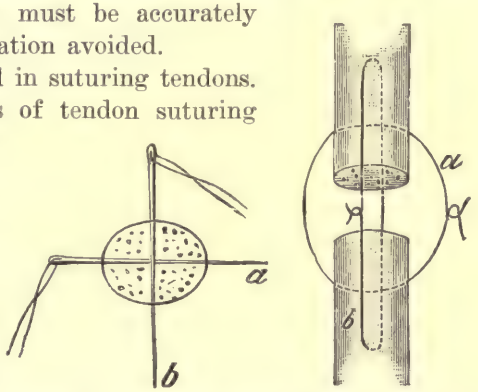


FIG. 213.—TENDON SUTURE ACCORDING TO FRIEDRICH. The first suture (*a*) is grasped by the second suture (*b*) which is passed parallel to the long axis of the tendon. After both sutures are applied the suture (*a*) is tied and then the suture (*b*). (Von Bergmann's "Handbook of Practical Surgery.")

The tendon sheaths, if opened by the vulnerating force or incised in searching for the tendons, should be closed over the tendons with fine catgut or silk sutures. Bands which retain the tendons in position, such as the anterior and posterior annular ligaments and the vincula, should be carefully sutured.

When a number of tendons have been sutured, adhesions may form between at the points of suture. Lotheisen has recommended that each

tendon be surrounded, after suture has been performed, by a sterilized gelatin tube in order to prevent these adhesions. [A flap of fat may be dissected upon and placed around the tendon when the wound is closed. Adhesions may be prevented in this way.]

The wound in the skin should then be sutured, an opening being left for drainage. Drainage should not be made directly over the tendons, as adhesions may then form between the tendons and the skin. If infection occurs, several sutures should be removed and iodoform gauze inserted. If a phlegmon develops all the skin sutures should be removed. It may then even be necessary to remove the sutures in the tendon sheaths.

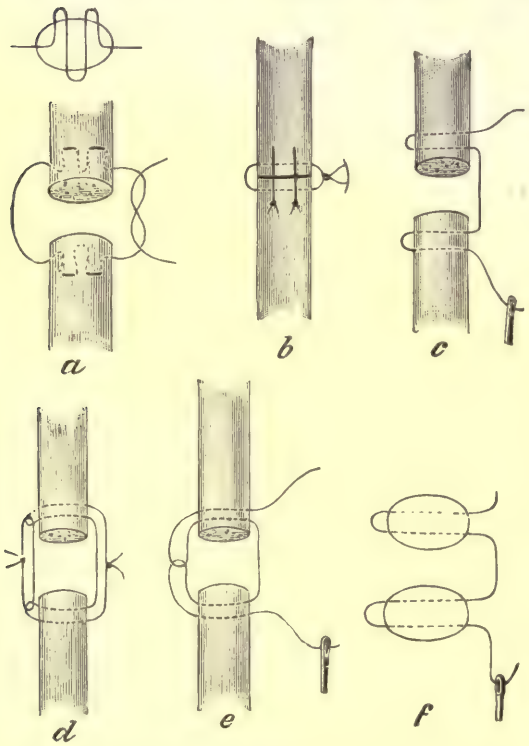


FIG. 214.—*a*, TENDON SUTURE ACCORDING TO WOELFLER; *b*, ACCORDING TO HAEGLER (AFTER THE FIRST SUTURE WHICH IS PASSED AT RIGHT ANGLES TO THE LONG AXIS OF THE TENDON IS TIED, IT IS GRASPED BY TWO SUTURES PASSED PARALLEL TO THE LONG AXIS OF THE TENDON); *c–f*, ACCORDING TO TRNKA.

After the ends have been united, the joint should be immobilized in such a position that there is no traction upon the sutures. If the tendons are small, immobilization should be continued for about four weeks; if large—for example, as large as the tendo Achillis and the ligamentum patellæ—it should be continued for at least six weeks.

Active and passive motions should be performed very cautiously when begun, as the new tissue which is formed in the repair of tendons is fragile. Warm baths and massage of the muscles involved are of great value. If the wound heals by primary union, a good result can usually be obtained after two or three months.

Tendons are repaired by the formation of scar tissue. This tissue is formed by the proliferation of the cells of the tendons and tendon sheaths, and by those of the surrounding connective tissue. These cells infiltrate the blood clot which forms between the divided ends of the tendons, and eventually become transformed into tissue which cannot

be differentiated after three months from that composing the tendons (Lit. by Marchand, Seggel).

Tenoplasty and Tendon Transplantation.—Two operative procedures —tenoplasty and tendon transplantation—may be employed to repair or overcome extensive defects in tendons. These procedures may also be used to correct contractures and overcome functional defects associated with paralysis.

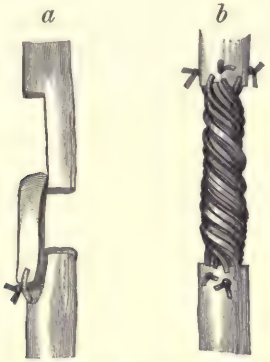


FIG. 215.—*a*, HUETER METHOD, SINGLE FLAP; *b*, GLÜCK'S METHOD, CATGUT REPAIR.

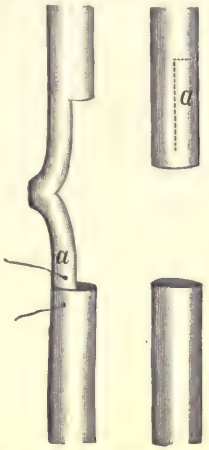


FIG. 216.—SINGLE-FLAP METHOD.

[" Tenoplasty (tendon lengthening) may be utilized to remedy deformities due to otherwise irremediable shortening of tendons dependent on contraction and sloughing, which are often the sequel of traumatism and inflammation.

" A tendon may be lengthened by a single flap (Figs. 215, *a*, 216) or it may require for the purpose the union of double flaps, one from the end of each extremity (Fig. 217).

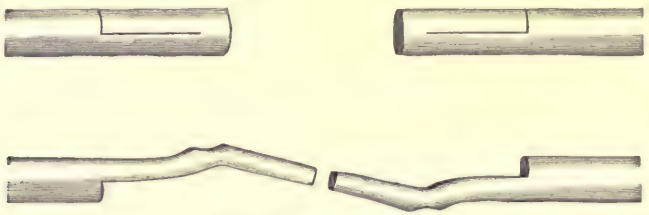


FIG. 217.—DOUBLE-FLAP METHOD.

" The making of alternate free incisions at the borders of a tendon (the accordion plan) so as to cause the tendon to assume an accordion appearance when lengthened (Fig. 218) is much more ingenious than practical. Less pronounced cutting (Fig. 219) followed by tendon lengthening is called the incision method (Fig. 220)."—Bryant's " Operative Surgery," Vol. I, p. 341.]

The transplantation of tendons was first attempted by Nicoladoni. It has been perfected by von Drobnik, Vulpius, Hoffa, and Lange.

The purpose of this operation is to transfer a part of the proximal end of a healthy muscle to the tendon of a paralyzed muscle or to the point of attachment of the latter. The flap taken from the healthy muscle naturally maintains its connection with it. This operation is intended to correct functional disturbances following injuries or diseases of tendons, those peculiar to muscular atrophy, and diseases of muscles and nerves.

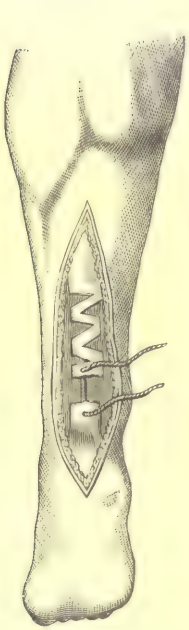


Fig. 218.

Fig. 219.

Fig. 220.

Fig. 218.—A. Poncet's Accordion Method.
Fig. 219.—Incision Method.
Fig. 220.—Tendon Lengthened in Incision Method.

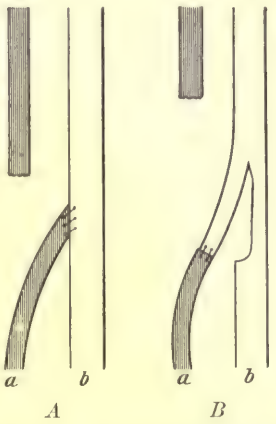


Fig. 221.—Lengthening of Tendons. In *A* the peripheral end of the defective, paralyzed or cut tendon is sutured into the side of a healthy adjacent tendon, while in *B* a flap is prepared from the healthy tendon into which the peripheral stump of the diseased tendon is sutured.

In this operation the distal end of the defective or paralyzed tendon which has been previously divided or vivified is sutured to the side of the tendon of a healthy muscle or to a flap taken from the tendon (Fig. 221). For example, the distal end of a divided flexor or extensor tendon of a finger may be sutured to one of the adjacent tendons which is intact.

Periosteal tendon transplantation is a modification of the preceding method. The distal end of the paralyzed tendon is not utilized, but a tendon, the function of which can be spared, or a flap from an adjacent tendon, is attached to the periosteum at the point normally occupied by the paralyzed one. The flap or the tendon which is transplanted is

passed beneath the fascia, a restraining band thus being formed for it. As in tenoplasty, a bridge of silk or of strands of catgut can be used to fill in the defect between the tendon and its periosteal attachment.

An immobilizing dressing of plaster-of-Paris must be applied after tendon transplantation, the part being immobilized in a position in which the affected tendons are relaxed. Immobilization should be continued for from five to seven weeks, after which time careful active and passive motion may be instituted.

LITERATURE.—*Becker.* Beiträge zu den traumatischen, nicht komplizierten Luxationen der Extensorensehnen der Finger. Münch. med. Wochenschr., 1903, No. 12.—*Blauel.* Ueber die Naht bei subkutaner Zerreissung des Lig. patellae.—Beitr. z. klin. Chir., Bd. 29, 1901.—*Borst.* Ueber die Heilungsvorgänge nach Sehnenplastik. Ziegler's Beitr. z. pathol. Anat., Bd. 34, 1903.—*Friedrich.* Subkutane Zerreissungen der Sehnen an Hand und Fingern. Handb. d. prakt. Chir., IV. Bd., 2. Aufl., p. 300.—*Haberern.* Ueber Sehnenluxationen. Deutsche Zeitschr. f. Chir., Bd. 62, 1902, p. 192.—*Hägler.* Ueber Sehnenverletzungen an Hand und Vorderarm. Beitr. z. klin. Chir., Bd. 16, 1896. *Kirchmayr.* Zur Kasuistik der subkutanen Sehnenrupturen. Wien. klin. Wochenschr., 1900, No. 45.—*Krukenberg.* Lehrbuch der mechanischen Heilmethoden. Stuttgart, 1896, pp. 72, 73.—*Küttner.* Ueber subkutane Sehnenscheidenhämatome. Beitr. z. klin. Chir., Bd. 44, 1904, p. 213.—*Lange.* Weitere Erfahrungen über seidene Sehnen. Münch. med. Wochenschr., 1902, No. 1.—*Lessing.* Traumatische subkutane Ruptur einer Fingerbeugersehne in ihrer Kontinuität. Beitr. z. klin. Chir., Bd. 30, 1901.—*Lotheisen.* Zur Technik der Sehnen- und Nervennaht. Arch. f. klin. Chir., Bd. 64, 1901;—Ueber die Zerreissungen im Streckapparate des Kniegelenkes. Beitr. z. klin. Chir., Bd. 24, 1899, p. 673.—*Marchand.* Der Prozess der Wundheilung. Deutsche Chir., 1901, Heilung der Sehnenwunden, p. 261.—*Schürmayer.* Luxation der Strecksehne des Mittelfingers. Zeitschr. f. Chir., 1897, p. 846.—*Seggel.* Histologische Untersuchungen über die Heilung von Sehnenwunden und Sehnendefekten. Beitr. z. klin. Chir., Bd. 37, p. 342, 1903.—*Witzel.* Ueber Sehnenverletzung und ihre Behandlung. v. Volkmann's Samml. klin. Vorträge, No. 291, 1887.

IV. INJURIES OF TENDON SHEATHS AND BURSÆ

Blood may be extravasated into tendon sheaths and bursæ after contusions and lacerations. A tense, frequently fluctuating, painful, circumscribed swelling which corresponds in position and size to the sheath or bursa involved then develops. The pain is increased when attempts at movement are made. Usually all the symptoms subside after a few weeks if the part is kept quiet and a bandage exerting mild compression is applied.

If the case is neglected a chronic inflammation develops and a hygroma forms. A similar condition is produced by foreign bodies if lodged in a tendon sheath or bursa and by repeated traumatism.

Open injuries of tendon sheaths and bursæ are very apt to be complicated by infection. If infection does develop in a tendon sheath, it may extend very rapidly to neighboring structures.

Aseptic treatment of the wounds and early incision of inflammatory foci are the indications to be met in the treatment of these cases.

V. INJURIES OF PERIPHERAL NERVES

Injuries of peripheral nerves may so interfere with or destroy their power of conduction that their function (transmission of sensory, motor, reflex, vasomotor, and trophic impulses) may be more or less completely interfered with. The degree of the functional disturbance depends for the most part upon the extent of the changes in the nerve fibers. Slight transitory disturbances follow destruction of the medullary sheaths; severe and permanent ones, injury and degeneration of the axis cylinders.

(a) SUBCUTANEOUS INJURIES

The most common subcutaneous injuries are concussions, contusions, lacerations, and subluxations. A concussion may affect a nerve trunk (for example, concussion of the ulnar nerve caused by a blow on the elbow) or all of its terminal fibers of distribution (for example, concussion associated with a fall, the patient alighting on the hands or feet). The transitory functional disturbances accompanied by pain disappear after a few minutes. A severe concussion of sensory nerves may produce the clinical picture of shock.

Contusion of Nerves.—Pressure palsy represents the mildest form of a nerve contusion. It frequently occurs when the upper or lower extremity is held in an improper position during sleep, the radial, ulnar, and sciatic nerves being most frequently involved. A sensation of numbness is first experienced. When the position of the limb is changed a tingling sensation is experienced for a short time, and then the normal conditions are rapidly restored. A paralysis which may last for several weeks may occur during anæsthesia if the arm is allowed to hang over the edge of the table and pressure is exerted upon the musculospiral nerve, or when the arm is elevated and abducted and the head of the humerus is pressed against the brachial plexus. Contusion of the sciatic nerve, followed by paralysis, is occasionally caused by attempts at reduction of traumatic and congenital dislocations of the hip. An Esmarch constrictor improperly applied or allowed to remain for too long a time may produce a long-standing or permanent paralysis, the radial, ulnar, and external popliteal nerves being most frequently injured in this way. Pressure exerted by callus is not an infrequent cause of an injury of this type. Nerves which are closely applied to bone, such as the musculospiral in the arm, the ulnar behind the internal epicondyle, and the external popliteal as it winds about the head of the fibula, are the ones most commonly affected. A complete paralysis which is preceded by

sensory disturbances and neuralgic pains develops, unless the nerve involved is dissected out of the callus. The same symptoms are produced when a nerve is imbedded in a mass of scar tissue, when it is pressed upon by a projecting fragment of a bone, or displaced and infiltrated by a new growth.

A sudden mild contusion may produce much the same clinical picture as a concussion, but a severe injury produced by blunt force, such as is associated with a blow or fall or is experienced when a fragment of a fractured bone is driven into the soft tissues, may be accompanied by marked functional disturbances. These may subside if conditions favorable for repair are provided, or may grow worse and become permanent if improperly treated.

Stretching and Laceration of Nerves.—Stretching and laceration of a nerve may be followed by transitory or permanent changes. Paralysis of the nerves of the arm may follow a dislocation of the shoulder. Disturbances of vision, loss of sensation, and paralysis of one half of the face may be caused by laceration or contusion of the optic, trigeminal, and facial nerves respectively in fractures of the base of the skull.

The recognition of nerve injuries of this type, as well as of those associated with open injuries, is usually based upon impairment of function of the nerves involved and upon the location of the wound.

Injuries of muscles, ischæmic palsies and contractures following injuries and inflammatory processes in the extremities may cause some difficulty in making a diagnosis. They are differentiated from nerve injuries by the absence of disturbances of sensation and of the reaction of degeneration.

The amount of restoration of function depends upon the extent of injury to the nerves, upon their ability to regenerate, and upon whether conditions favorable for regeneration are provided.

The treatment of simple subcutaneous contusions and lacerations of nerves should be conservative. During the first week the part involved should be immobilized in a cast, in order to prevent any added injury to the nerves by movements. When electrical treatment is instituted later, the weak galvanic current should be employed, if there is a reaction of degeneration. The cathode should be placed over the seat of injury, the anode over the plexus (Oppenheim).

A complete division of the nerve can be excluded if the loss of function is only partial. Complete restitution of function occurs after a few weeks in these cases if a conservative line of treatment is followed. If the injury apparently involves the entire diameter of the nerve and no results followed electrical treatment continued

from four to six weeks, operative interference should no longer be delayed. After this time but little can be expected of conservative treatment, and if the operation is delayed the prognosis becomes bad. When the nerve is exposed it is usually found imbedded in a mass of scar tissue. If the nerve is completely severed, its ends are separated by it. After the ends of the nerve have been dissected free, they should be vivified or resected and sutured or united by neuroplasty.

If the paralysis is due to the inclusion of the nerve in a callus or a mass of scar tissue, it should be dissected out of the mass enclosing it (neurolysis). In order to prevent pressure from scar tissue developing after neurolysis, the nerve should be surrounded by a flap of muscle or by one made from the surrounding fatty tissues. If the electrical conductivity of the nerve has not been lost, its function is usually restored within a few days. Even when there is doubt as to the condition of a nerve, neurolysis should first be tried, and then if there is no return of function after a number of weeks, the injured area should be resected and the ends sutured (Schede).

Subluxation of Nerves.—The ulnar and external popliteal nerves are about the only ones that are ever displaced. Displacements of these nerves are most often associated with fractures of the medial epicondyle of the humerus and of the head of the fibula. If the medial epicondyle is very small and flat and the ulnar nerve is not firmly fixed in its groove, it may be displaced by sudden forcible flexion of the forearm. An habitual displacement which may be either congenital or traumatic may even occur. In such a case the nerve is partially or completely displaced with every attempt at flexion of the forearm and when the triceps muscle contracts forcibly, but the patient experiences no pain or disturbance of function (Momburg).

Symptoms, as a rule, develop only when the displacement is due to traumatism. They are then due to a contusion or inflammation of the nerve. A positive diagnosis can be made of the nature of the lesion by pain which radiates throughout the entire distribution of the nerve, by sensory and motor disturbances, and by finding the painful nerve in an abnormal position.

If there are no functional disturbances associated with the displacement, treatment is not necessary. If the displaced nerve is painful or its function is interfered with, it should be dissected free, replaced in its normal position, and maintained there by suturing a flap of fascia or muscle over it (Momburg). If the ulnar nerve is but recently displaced, an attempt should be made to reduce it without an operation. If the attempt is successful, the forearm should be immobilized in the extended position until repair is completed.

(b) OPEN INJURIES OF NERVES

In open wounds nerves may be partially or completely cut across, pierced, shot through, crushed, or lacerated. Crushing and lacerating injuries of nerves are most frequently associated with complicated fractures, the displaced fragments of bone injuring the nerves. Fragments of bone, likewise foreign bodies (pieces of glass, wood, and metal, bullets and fragments of shells) may become lodged in a nerve and surrounded by a dense connective tissue. This scar tissue gradually contracts until the function of the nerve, which was at first merely impaired, is completely destroyed. Occasionally a ligature is applied to a nerve by mistake, marked functional disturbances developing as a result. Large segments of nerves may be torn away in injuries associated with explosions of boilers and shells, in machinery accidents, and in bites inflicted by wild animals.

The divided ends of a nerve retract but little unless they are displaced by fragments of bone or lacerated muscles. The cut surface never appears smooth as the nerve bundles project beyond it. A contused area may be recognized directly after the injury by its red color and soft consistency.

Degeneration of Divided Nerves.—Degeneration follows the separation of nerve fibers; it makes no difference whether they are cut across, lacerated, or contused. A limited traumatic degeneration occurs very soon at the point of injury. This is followed in from two to four days by the secondary (paralytic) degeneration which extends to all that part of the nerve distal to the point of injury, and also involves a small segment immediately proximal to the wound. Therefore a direct union of the nerve ends with restitution of function (the so-called *prima intentio nervorum*) is an impossibility even when the connections between the nerve fibers are retained or the cut surfaces are immediately approximated and sutured. Degeneration of injured nerves always occurs. It is the primary process which ends in the regeneration of fibers and the repair of nerves.

Regeneration of Nerves.—The beginning of regenerative changes is indicated as early as the second day by enlargement of the nuclei of the cells of the sheath of Schwann and numerous karyokinetic figures. Degeneration of the medullary sheath is indicated by the accumulation of fragments, balls, and granules of myelin; of the axis cylinders by fibrillation. The formation of new axis cylinders and medullary sheaths is apparently associated with the regenerative changes in the cells of the sheath of Schwann (von Buengner, P. Ziegler, Wieting, Marchand). There is, however, no uniformity of opinion among investigators as to the rôle played by the sheath cells in the regeneration of nerves, many

(Ranvier, Vanlair, von Notthaft, Stroebe) believing that new axis cylinders are formed only by outgrowths from preëxisting ones.

The new nerve fibers are first found in the proximal end of the divided nerve. They are connected with the old fibers. In favorable cases these new fibers infiltrate the granulation tissue which is formed from the sheath cells and bridges over the space between the divided ends. These newly formed fibers then gradually grow into the distal segment of the nerve even to its terminal filaments. Waller, Vanlair, Stroebe, and others believe that this so-called neurotization is the method by which nerves regenerate. Von Buengner, Wieting, and Kennedy believe that the fibers which develop in the proximal end form a direct union with incompletely differentiated elements in the distal segment, and that the latter go on to complete differentiation only after this union is complete.

Conditions Favoring and Retarding Regeneration.—Absence of wound infection, incomplete division of a nerve (or after complete division accurate approximation of the cut ends), and very limited injuries, such as might be produced by tying a ligature about a nerve, are the conditions which favor most complete repair.

Repair may be retarded or prevented if an infection develops, if a large amount of scar tissue develops between the divided ends, if a large segment of the nerve is destroyed, or if, after complete division, muscle, bone, or a foreign body becomes interposed between the ends or the latter are displaced and remain out of line.

If any of these conditions are present the proximal end becomes bulbous (amputation neuroma), while connective tissue develops in the distal portion of the nerve, which gradually decreases in size following the absorption of the fragmented myelin.

Secondary Changes in Muscles.—The muscles supplied by the injured nerve eventually undergo complete atrophy (neurogenous muscular atrophy).

Motor and mixed nerves have but little regenerative power, and it cannot be definitely said that repair will always follow even when favorable conditions are provided. This is so, notwithstanding the fact that it has been demonstrated by animal experiments that defects several centimeters in length may heal spontaneously (Tiedemann), or after bridging over the space between the divided ends by using decalcified bone tubules (Vanlair), sterilized segments of arteries (von Buengner); strands of catgut (Gluck, Assaky), pieces of nerves from other animals (Gluck), and that spontaneous union (e. g., after laceration of the brachial plexus by a fragment of a bomb, Langenbeck) and union after the use of the devices mentioned above have also occurred in man. Regeneration occurs much more frequently and completely in sensory

nerves, as the return of neuralgic pains after extensive removal of the branches of the trigeminal nerve and the restoration of sensation in the skin of the neck after the radical removal of tuberculous lymph nodes indicate.

Symptoms.—The symptoms of a nerve injury are indicated by loss of function and develop immediately. The symptoms depend upon the function of the nerves, and after injury of mixed nerves, which are most frequently affected, they are both sensory and motor. Eventually vasomotor and trophic disturbances are also noted. Pain and paræsthesia are most commonly associated with incomplete division of sensory and mixed nerves.

Diagnosis.—The diagnosis is based upon the anatomical position of the wound or scar, and upon the presence of motor and sensory disturbances.

The motor symptoms consist of a flaccid paralysis, absence of or impaired reflexes, rapidly developing muscular atrophy, and loss of function of the muscles supplied by the injured nerves. The disturbances of function following the injuries of nerves such as the musculospiral, ulnar, external popliteal, facial, etc., are very characteristic. The diagnosis may be quite difficult if but a single muscle is affected as a result of an injury to a nerve or plexus, as the loss of function may be compensated by synergists.

Reaction of Degeneration.—The electrical reaction of the nerves and the muscles supplied by them should be determined in all cases in which an injury to a nerve is suspected. The reaction of a divided nerve to faradic and galvanic stimulation gradually decreases in intensity and rapidity, disappearing completely after twelve days. The reaction of muscles supplied by the divided nerves to the faradic current disappears in the same way. Within two weeks, however, the reaction of the degenerating muscle to the galvanic current is intensified, and the reaction of degeneration develops—the A.C.C. being greater than the C.C.C. This reaction becomes most marked in from three to four weeks, and then persists for months. Sometimes this increased irritability to the galvanic current does not disappear until after a year, when the atrophy of the muscles is complete.

When degeneration occurs the antagonistic muscles gradually contract, producing deformities. Contractures of the different parts are produced in this way.

Sensory Disturbances.—The sensory disturbances following injuries of sensory and mixed nerves do not extend over the entire area supplied by the nerves involved. The terminal filaments of these nerves usually anastomose freely, so that nerves supplying adjacent cutaneous areas are united (e. g., anastomoses between the musculocutaneous and

median nerves), and besides there is an overlapping of the cutaneous fields, many nerves sending branches into the same area. As a result of these peculiarities in the distribution of sensory and mixed nerves, the sensory disturbances are usually limited to a small area which gradually decreases in size even when there is no regeneration of the injured nerves, for filaments grow into the area from branches of adjacent nerves. Permanent and extensive sensory disturbances occur only after injury of many adjacent nerves or of all the cords of a plexus (Kölliker, Oppenheim, Schede).

Vasomotor and Trophic Disturbances.—Vasomotor disturbances are indicated by redness, cyanosis, and a lowering of the temperature of the skin.

Trophic disturbances affect usually the skin and its appendages. The skin becomes glossy and smooth; there is a tendency to eczema. Herpes zoster and ulcers develop and the parts become atrophic. The bones may become atrophic and growth may be retarded. Sometimes a serous exudate which is transitory and is followed by stiffness is poured out into the joints.

Special symptoms follow division of the sympathetic, vagus, phrenic, and cranial nerves. These are most often injured in gunshot and stab wounds of the neck and head, or during operations. The symptoms and results of injuries of these nerves are discussed in special surgeries.

Neuralgia and chronic neuritis should also be mentioned as some of the remote results of nerve injuries. Neuralgia not infrequently develops when a nerve has been exposed to pressure or repeated traumas, when it has been partially severed, contains a foreign body, or when, after regeneration, it becomes so attached to surrounding tissues by a scar that it is pulled upon whenever a movement is attempted. Neuritis may develop if infection occurs in an open wound or if a foreign body or an inflammatory exudate is situated close to a nerve. Neuritis rarely develops after a simple contusion.

Treatment.—In recent cases of injuries to nerves associated with open wounds the treatment should be operative. The wound and the area surrounding it should be carefully sterilized, a constrictor applied, and an attempt made to find the ends of the divided nerve. When found they should be carefully approximated by sutures. Primary nerve suture should also be attempted in large contused and lacerated wounds, unless the defect in the nerve is so great as to render it impossible. A conservative expectant treatment is to be recommended only after gunshot wounds, especially after wounds caused by small projectiles. Usually in these cases the nerve is not completely divided, its conductivity being merely interfered with for a short time, and recovery occurs spontaneously. If, in cases of this character, there is

no improvement of function after several weeks, the nerve should be exposed at the seat of injury, the scar tissue removed, the vivified ends of the nerve sutured, and foreign bodies which are frequently present removed. This is called secondary nerve suture.

Nerve Suture.—In performing nerve suture in old cases, the divided ends should first be vivified; the lacerated, contused ends, or central end, which may have become transformed into a neuroma, being removed with a sharp knife and converted into a smooth surface.

Scissors should not be used to vivify the ends as they crush the fibers. The vivified ends should then be approximated, the most careful asepsis being practiced, and tension upon the ends being avoided. It is advisable to employ the so-called direct suture, some fine catgut sutures being passed through the ends of the nerve. In suturing small nerves it may be necessary to pass the suture through the entire thickness of the nerve involved. In suturing larger nerves the sutures should grasp merely the outer part of the nerve trunk, avoiding in this way injury of any great number of fibers. A round, non-cutting needle should be used. In the indirect nerve suture recommended by Hueter, injury of the nerve fibers is entirely avoided, but an accurate approximation of the ends not secured. In this method only the paraneural connective tissues are grasped in the sutures. It is advisable to combine both the direct and indirect methods (Tillmanns). This is the method of nerve suture employed in the von Bergmann clinic. In suturing very delicate nerves it is often necessary to employ the paraneural suture alone. Some of the other procedures which have been employed in uniting nerves should be mentioned. In order to avoid adhesions between the line of suture and surrounding tissues, which often cause neuralgia and marked functional disturbances, Payr has recommended that the suture line be protected by a magnesium tube. Lotheisen recommends that the nerve be imbedded in a gelatin tubule hardened in formalin. Foramitti recommends that a segment of an artery or vein taken from a calf and thoroughly sterilized be used for the same purpose. The magnesium tube is first passed over the end of the nerve, and after the suturing is completed it is slipped over the suture line. The gelatin tubule or the segment of an artery or vein may be split and placed over the nerve after the suturing is completed. These procedures are now used instead of the one devised by Vanlair, in which a decalcified piece of bone was used to bridge over the defect.

Von Bruns has recommended in secondary suture that a longitudinal incision be made in the scar tissue uniting the ends and that this incision should then be sutured transversely, hoping in this way to approximate the ends of the nerve fibers (Fig. 222). When there is no bridge of scar tissue, he has recommended that a V-shaped piece

should be cut out of the bulbous central end, and that the distal end should be sutured into it after it has been given the proper shape (Fig. 223).

It is especially difficult to unite divided ends of nerves when there is a wide interval between them resulting from a laceration or contusion

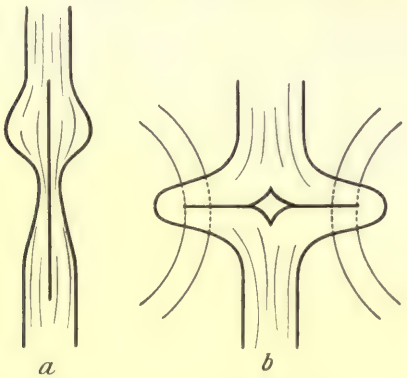


FIG. 222.—SECONDARY NERVE SUTURE ACCORDING TO VON BRUNS. *a*, Longitudinal incision in the scar in the nerve, which in (*b*) is sutured transversely.

FIG. 223.—SUTURE OF THE POINTED PERIPHERAL END OF THE NERVE INTO THE PROXIMAL END.

of a long segment, from the resection of a mass of scar tissue or the removal of a tumor.

Even when there is quite a defect nerve suture should be attempted, as it is the most reliable procedure. Small defects up to 3 cm. in length can be repaired without tension by cautiously drawing the ends together. Greater defects, even up to 8 cm. in length, in some of the larger nerves (radial, median, sciatic, and popliteal) may be repaired by placing the joint in extreme flexion, extension, or other position demanded by the case, and by maintaining it in this position for a number of weeks. When union is probably very well established the part should gradually be returned to its normal position. If a long segment of a nerve has been destroyed, a piece of bone may be resected, an actual shortening of the part being produced in the way which permits of approximation of the ends of the nerve (von Bergmann, Loebker, and others).

Neuroplasty and nerve grafting are not as reliable as nerve suture. They may be employed when the latter is technically impossible.

Neuroplasty and Nerve Grafting.—By neuroplasty, a procedure first employed by Létiévant, is understood the repair of defects in nerves by the use of flaps, the latter being made from the proximal, distal, or both ends of the divided nerve. The bridging over of a defect by a flap made from the divided ends, which are united by sutures, may be

followed by a complete restoration of function (Tillmanns). Other methods in which the defect is filled in with some foreign material, which acts as a tunnel or scaffolding for the developing fibers, are also successful. Decalcified tubules of bone (Vanlair's tubulization), strands of catgut (Gluck, Assaky), and segments of nerves taken from other animals (Gluck, Landerer) have been used for this purpose.

The procedure known as nerve grafting was also devised by Létiévant. In this method the distal end of the injured or paralyzed nerve is sutured to some adjacent uninjured nerve. The distal end of the injured nerve may be sutured to the side of the uninjured nerve, which has been vivified or, as has been suggested by Deprés, into a slit which is made in the latter. The attachment of the distal end to the side of the uninjured, even when the latter has not been vivified, is sometimes successful (Manasse). [No definite results can be expected unless the nerve is vivified and the last-mentioned method should not be employed.] The distal end of an extensively lacerated median nerve has been successfully united with the ulnar (Deprés). The following successful cases of nerve grafting may be mentioned: Grafting of the distal end of the ulnar to the median; of the distal end of the injured musculospiral to a flap made from the median (Sick); of the distal end of the paralyzed facial to the spinal accessory (Faure and Furat, Hackenbruch, Cushing, Ballance and Stewart), or to the hypoglossal (Koerte, Frazier).

Care of the Cutaneous Wound and Dressing.—When a secondary nerve suture is performed the wound in the skin can always be closed by a plastic operation or by skin grafting. In primary suture the wound should not be closed unless the conditions are favorable for repair. If the wound must be left open, the nerve should be surrounded with a flap of muscle or fat in order to prevent the scar from pressing upon it.

After the wound is dressed, the part should be immobilized in a splint or plaster-of-Paris dressing in a position which permits of no tension upon the sutured ends. Immobilization should be continued from three to four weeks. When the immobilizing dressing is removed, the return of function may be hastened by electricity, massage, active and passive motion.

Restoration of sensation is the first indication that a nerve suture has been successful. This, however, may be deceiving, as the return of sensation may be due to the overlapping of cutaneous sensory fields or to the growth of filaments from adjacent nerves. Then the reaction of degeneration disappears, the changes incited by the galvanic and faradic currents approaching the normal. The return of motion is first indicated by muscular twitching when an attempt is made to throw the muscle into action; later by fairly powerful contractions.

Time Required for the Restoration of Function.—The time required for complete return of function differs. Function is usually reëstablished more quickly after primary than after secondary nerve suture as the muscular atrophy which occurs when the divided ends are not sutured early becomes so extensive that muscular activity is restored slowly. The nearer the periphery the point of suture the more quickly regeneration occurs (Etzold). A long time is required after neuroplasty for complete restoration of function.

Sensation may return in from two to four weeks if a nerve suture is successful. A longer time is required for the return of motion. It never returns earlier than three weeks; usually in about six weeks; a year or more may pass before motion returns.

Results of Nerve Suture.—[The results of nerve suture are flatteringly exhibited in the following table (Bowlby):

	Successful	Improved	Failure	Total
Primary suture.....	32	34	14	80
Secondary suture.....	32	26	15	73

These statistics would seem to indicate that the results following secondary suture are about as good as those following primary suture. —Bryant's "Operative Surgery," Vol. I, p. 274.] A nerve suture may be successful even when undertaken a number of years after the nerve was divided. Tillaux successfully sutured the median nerve fourteen years after division.

If the suture is not successful, the nerve should be exposed at the point of union, and the scar tissue, which is often to blame for the failure, dissected away. If the ends have become separated by scar tissue, the latter should be removed and the vivified ends again sutured.

LITERATURE.—*Ballance* and *Stewart.* On the Operative Treatment of Chronic Facial Paralysis of Peripheral Origin. British Medical Journal, 1903. Hildebrands Jahresber., 1903.—*Cushing.* The Surgical Treatment of Facial Paralysis by Nerve Anastomosis. Annals of Surgery, 1903. Hildebrands Jahresber., 1903.—*Dumstrey.* Ueber Nervenpfropfung. Deutsche Zeitschr. f. Chir., Bd. 62, 1902, p. 40.—*Etzold.* Ueber Nervennaht. Ebenda, Bd. 29, 1889, p. 430.—*Foramitti.* Zur Technik der Nervennaht. Arch. f. klin. Chir., Bd. 73, 1904.—*Frazier* and *Spiller.* The Surgical Treatment of Facial Paralysis. Univ. of Pennsylvania Med. Bulletin, 1903.—Hildebrands Jahresber., 1903.—*Gleiss* (v. Bruns). Beiträge zur Nervennaht. Beitr. z. klin. Chir., Bd. 10, 1893, p. 387.—*Hackenbruch.* Zur Behandlung der Gesichtslähmung durch Nervenpfropfung. Chir. Kongr.-Verhandl., 1903, II, p. 231.—*Haim.* Ueber Luxation des Ulnaris. Deutsche Zeitschr. f. Chir., Bd. 74, 1904, p. 96.—*Th. Kölliker.* Die Verletzungen und chirurgischen Erkrankungen der peripheren Nerven. Deutsche Chir., 1890.—*Körte.* Ein Fall von Nervenpfropfung des N. facialis auf den N. hyperglossus. Deutsche med. Wochenschr., 1903, No. 17.—*Kramer.* Zur Neurolyse und Nervennaht. Beitr. z. klin. Chir., Bd. 28, 1900, p. 423.—*Lotheisen.* Zur Technik der Nerven- und Sehnennaht. Arch. f. klin. Chir., Bd. 64, 1901, p. 310.—*Marchand.* Der Prozess der Wundheilung.

Deutsche Chir., 1901, Heilung der Nervenwunden, p. 335.—*Momburg.* Die Luxation des N. ulnaris. Arch. f. klin. Chir., Bd. 70, 1903.—*Oppenheim.* Lehrbuch der Nervenkrankheiten, Berlin.—*Payr.* Zur Technik der Blutgefäss- und Nervennaht (Magnesiumprothese). Chir. Kongr.-Verhandl., 1900, II, p. 593.—*Riethus.* Ueber Verletzungen des N. radialis bei Humerusfrakturen und ihre operative Behandlung. Beitr. z. klin. Chir., Bd. 24, 1899, p. 703.—*Schede.* Chirurgie der peripheren Nerven und des Rückenmarks. Handb. der spez. Therapie der inneren Krankheiten von Penzoldt und Stintzing, 1898.—*Schütte.* Die Degeneration und Regeneration peripherer Nerven nach Verletzungen. Zusammenfassendes Referat mit Lit. Zentralbl. f. Pathol., 1904, Bd. 15, p. 917.—*Tillmanns.* Ueber Nervenverletzungen und Nervennaht. Arch. f. klin. Chir., Bd. 27, 1882.—*P. Ziegler.* Untersuchungen über die Regeneration des Achsenzylinders durchtrennter peripherer Nerven. Ebenda, Bd. 51, 1896, p. 796.

VI. INJURIES OF BLOOD VESSELS

Subcutaneous injuries of the larger blood vessels may be associated with contused and lacerated wounds. Naturally normal vessels are more resistant to trauma than are diseased vessels, the walls of which have been weakened by arteriosclerosis or thinned by suppuration or by pressure exerted by tumors.

Injuries of the vessels may be associated with fractures and dislocations, or inflicted when attempts are made to reduce old dislocations. They may also be caused by different forms of trauma.

The immediate and remote results of an injury to a vessel depend not only upon the character of the trauma, but also upon the resistance of the different tunics of the vessel involved. The intima is most susceptible to trauma. A relatively insignificant laceration or contusion such as is produced by a ligature may cause a destruction of the endothelium, a superficial or deep tear of the intima. A small thrombus composed of blood platelets then forms at the point of injury, the intima later proliferating to fill in the defect.

If the media of an artery is injured the resulting scar may gradually give way under the blood-pressure, a true traumatic aneurysm then developing. Either a mural or an obturating thrombus may form after severe contusions or lacerations of arteries and veins in which the intima is separated for some distance and rolls in. The angiotribe crushes the intima and media of vessels, without injuring to any extent the adventitia. The angiotribe is not employed very extensively at the present time, as the thrombus which forms after its use cannot be relied upon to check hæmorrhage. The thrombus is often displaced when the blood pressure rises as the patient is recovering from anæsthesia, and ligation is therefore preferred to angiotripsy.

Injuries of all the coats of a vessel may be followed by complete division of the vessel, or a wound not involving the complete circumference of the wall. The latter, accompanied by the extravasation of

considerable blood, may be closed first by a thrombus; later by scar tissue, providing a pulsating hæmatoma or false aneurysm does not develop. Crushed and lacerated vessels are often closed by curling up of the intima, so that there is no palpable or visible extravasation of blood.

(a) SUBCUTANEOUS INJURIES OF VESSELS

Subcutaneous injuries of the larger vessels occur most frequently in the extremities. The injuries are produced by fractured or dislocated bones or by some external violence. A vessel may be ruptured during attempts at reduction of an old dislocation. The character and extent of the injury vary a great deal in different cases. Abdominal injuries produced by blunt force (a blow or kick, being run over by a wagon) may be accompanied by rupture of mesenteric vessels, the latter being forced against the vertebræ. Fractures of the skull may be complicated by hæmorrhage from the middle meningeal artery, which proves fatal unless controlled. It is of medico-legal interest to know that the intima of the common carotid artery may be injured when a person is hanged or strangled by a rope.

Symptoms.—Symptoms develop in these subcutaneous injuries only when some important vessel bleeds or is closed by a thrombus. Small defects in the intima heal without giving rise to symptoms, or are recognized only when an aneurysm forms; for example, an aneurysm of the abdominal aorta following a subcutaneous injury of the abdomen. If a large amount of blood is poured out into the tissues surrounding a large artery shortly after a subcutaneous injury or fracture, the possibility of a tear or rupture of a large vessel must be considered. If a rough systolic murmur can be heard over the seat of injury, a diagnosis of some arterial lesion can usually be made with certainty. This systolic bruit, which is called von Wahl's sign, occurs after laceration of the intima in subcutaneous injuries, and is also associated with punctured and gunshot wounds of arteries. It is not present after injuries of small vessels (Rotter), and may be wanting after injuries of larger ones. The sign may be caused by a widening of the lumen when the wound gapes, or by a narrowing of the lumen when the intima is dissected up or a mural thrombus forms. A positive diagnosis of a complete rupture or closure by thrombus formation of the principal artery can be made if, after the injury, the extremity becomes cool, pale, or cyanotic, and the pulse can no longer be felt. Frequently the veins are also closed by thrombi when the extremity has been run over by some heavy vehicle or by a car.

Treatment.—An operation should be performed immediately if a rapidly enlarging hæmatoma forms. A hæmorrhage into the abdominal

or cranial cavities rapidly proves fatal unless controlled. A hæmorrhage into the tissues of an extremity may cause gangrene, as the pressure exerted by the extravasated blood prevents the establishment of a collateral circulation. If rupture of a vessel is suspected, an Esmarch constrictor should be applied, the hæmatoma incised, the blood clots removed with sterile gauze, and the injured point found. If the artery is essential to the life of the part or organ which it supplies, it should be closed by a lateral or an end-to-end suture, depending upon the character of the injury. If not essential, or the anastomosis between it and other vessels is very free, it should be ligated above and below the point of injury.

If a diagnosis of thrombosis of one of the large vessels of an extremity is made, the development of a collateral circulation should be favored by elevating the extremity and incising large hæmatomas.

(b) OPEN INJURIES OF LARGER BLOOD VESSELS

Injuries of arteries are not infrequently associated with incised, contused, punctured, and gunshot wounds; with extensive mutilating injuries caused by explosions, fragments of a shell, machines; with the tearing away of an entire extremity, and with complicated fractures and dislocations. Veins are injured more frequently than arteries, for they are firmly attached to the fascia and are not pushed aside by the vulnerating force as the arteries are. The walls of veins are thinner and less resistant to trauma than are the walls of arteries. All sorts of foreign bodies, such as pieces of metal, glass, and wood, needles, and points of knives, may lodge in or penetrate the walls of blood vessels. Blood vessels may also be injured during operations by a knife, scissors, sharp hook or spoon, or by the finger when a blunt dissection is made. An erosion of a vessel—which may be caused by the pressure of a fragment of bone, an improperly placed drainage tube, or a tracheal canula—should be classified with this type of wounds.

Injuries involving but a portion of the vessel wall are differentiated from those in which the vessel is completely divided.

Penetrating wounds of vessels, which are not infrequently associated with punctured, gunshot, and contused wounds, heal without giving rise to symptoms, unless an aneurysm (aneurysma traumaticum verum) forms.

Wounds that penetrate the vessel wall are followed by hæmorrhage, if the foreign body does not close the opening in the vessel and if the wound canal in the soft tissues is not closed by a clot or the interposition of muscles and fascia. The severity of the hæmorrhage depends upon the size of the wound and the caliber of the vessel. A small opening in a vein is rapidly closed by pressure exerted by extravasated

blood, and its lumen is later occluded by a thrombus. A pulsating hæmatoma (false aneurysm) usually develops after a penetrating wound of an artery. An arteriovenous aneurysm may develop if adjacent points in the walls of an artery and vein are injured simultaneously. Spontaneous closure by thrombus and scar formation after wounds of arteries occurs only when the wound, such as is produced by a needle, is narrow.

The results of the complete division of an artery differ markedly from those above mentioned. The artery gapes, if the division is clean-cut, and the hæmorrhage is severe, the amount of blood which is lost depending upon the size of the vessel, unless its lumen is closed by the contraction of the surrounding muscles or the cut vessel retracts from the margins of the wound.

Venous and Arterial Hæmorrhage.—Venous hæmorrhage is distinguished from arterial hæmorrhage by the color of the blood and the way in which it is discharged.

In arterial hæmorrhage the blood appears as a bright red stream, and is discharged in jets which correspond to the pulse beat. Blood discharged from veins is darker than that discharged from arteries, and the blood either wells up from the wound in the vein or is discharged in much weaker jets than is arterial blood. The amount of blood lost and the way in which it is discharged after injuries of veins depend upon a number of conditions. If the return flow is interfered with by a loosely applied constrictor or a dependent position of the limb, a great amount of venous blood is discharged. Special conditions favoring venous hæmorrhage are found in the anatomical relations at the base of the neck. The veins in this position are attached to the resistant deep cervical fascia, and do not collapse when injured. Besides, during expiration the venous blood is forced back into these veins, and blood is then discharged in jets; while during inspiration the blood is aspirated, and if there is a wound in the vein wall, air may be sucked into the circulation (air embolism). Aspiration of air into a vein is indicated by a gurgling sound. If air reaches the heart, symptoms of air embolism, which is frequently fatal, develop.

The characteristic differences between arterial and venous hæmorrhage as described above may be wanting if the wound is small or the outflow is interfered with. Naturally there is nothing characteristic about the hæmorrhage if an artery and a vein are injured simultaneously.

The results of a division of a vessel by blunt force, such as occurs in contusions and lacerations, differ from those following clean-cut division. If vessels are divided in a contusion or are lacerated, the intima and media curl up and occlude the lumen, or at least favor the development of an obturating thrombus. There may be no hæmorrhage

after laceration of even the very large vessels, and after severe injuries death from hæmorrhage is frequently prevented by this curling up of the intima and media. When an artery is put upon a stretch, the intima ruptures first, and then the media at a point nearer the periphery. These two coats then roll up and occlude the lumen, the adventitia being pulled out to form a thin band of tissue, just like a heated glass canula is drawn out to form a delicate capillary tube. If the artery is twisted at the same time that it is torn the closure is still more firm.

The dangers of open injuries to vessels are partly due to primary hæmorrhage, partly to secondary hæmorrhage and air embolism.

Secondary Hæmorrhage.—Secondary hæmorrhage occurs quite frequently after the primary hæmorrhage has subsided spontaneously. Secondary hæmorrhage occurs most frequently when the knife or other foreign body which may have closed the wound is withdrawn, when the thrombus which may have developed and closed the wound after an injury produced by blunt force is destroyed by suppuration or is dislodged by movements, or finally when the arterial wall becomes necrotic as the result of pressure exerted by a displaced fragment of bone.

Air Embolism.—The entrance of air into the injured internal jugular or subclavian vein during inspiration leads to the condition known as air embolism. In severe cases the patient dies immediately or after a few hours; the symptoms being great unrest, a feeling of anxiety, marked dyspnœa, cyanosis, weak, fluttering pulse, loss of consciousness, and convulsions. These symptoms are caused by interference with cardiac and respiratory functions, and by cerebral anæmia, for the aspirated air reaches the right heart and then passes into the pulmonary arteries interrupting the circulation. As a result of this occlusion of the pulmonary arteries the left heart does not receive enough blood to maintain the nutrition of the important centers in the brain and spinal cord. Other causes of death in air embolism, besides the sudden anæmia of the brain (Panum), are paralysis of the heart caused by dilatation of the right ventricle following the accumulation of air, and interference with the pulmonary circulation (Senn).

Mild cases of air embolism are observed after operations more often than after injuries. When air embolism occurs during an operation, but little air is, as a rule, permitted to enter the vein, as the condition is immediately recognized and the opening is closed by pressure. In these cases either no symptoms develop or, if they do, they are mild and transitory. Notwithstanding that the symptoms may be mild, an attempt should always be made to express the air, as it is impossible to estimate the amount which has been aspirated. The chest should be compressed during expiration, and during inspiration digital pressure

should be made over the opening in the vein in order to prevent the aspiration of more air (Treves). It is well in these cases either not to wipe away the blood which may have accumulated over the wound or to flood the field of operation with salt solution, preventing in this way the aspiration of air. When the danger of air embolism has passed, the vein should be seized with an artery forceps without teeth and ligated or sutured. In severe cases aspiration of the right ventricle may be indicated.

Diagnosis of Open Injuries.—The diagnosis of an open injury of a vessel is difficult only when the most important symptom—severe hæmorrhage—is wanting. A non-penetrating wound may escape notice when the vessel is not freely exposed in the wound. It is not unusual for the primary hæmorrhage to cease rapidly, and for a hæmatoma to fail to develop when the wound is made with a delicate fusiform instrument or by a projectile of small caliber. This is especially apt to happen if the vessel is covered by a thick resistant fascia or an aponeurosis (e. g., the femoral artery in Hunter's canal). In such cases the position of the wound offers the only clew to diagnosis, which is often later verified by the development of a bruit at the point of injury.

When a large hæmatoma develops after a small wound of a vascular region, it is not always possible to determine the nature of the injury to the vessel. If the pulse is wanting in the peripheral parts, the principal artery has probably been completely divided. If the pulse is present, but is weaker than on the uninjured side, and a systolic bruit which is transmitted toward the periphery is heard, one may conclude that the artery has received a lateral injury. If the bruit is transmitted both proximalward and distalward, one may conclude that a communication has been established between an artery and a vein, following simultaneous injury of adjacent parts of the walls of the vessels (arteriovenous aneurysm).

Treatment.—The treatment consists of temporary and permanent control of the hæmorrhage, precautions being taken against loss of blood and infection during the ligation or suturing of the injured vessels. If a diagnosis of an injury to an artery with or without simultaneous injury of a vein is made, the point of injury should be exposed, even if there is no external hæmorrhage, the blood clots removed, and the vessel ligated or sutured. The treatment becomes much more difficult if operation is delayed until an aneurysm has formed. A stab or gunshot wound of a vessel may heal with the development of but a small hæmatoma.

After ligation or suture of a large vessel an immobilizing dressing should be worn from two to three weeks.

If the hæmorrhage is severe the life of the patient often depends

upon the prompt and proper temporary control of the hæmorrhage. In the extremities it can be easily controlled by making pressure upon the principal artery proximal to the point of injury, or by constricting the extremity above the wound by a handkerchief, strap, piece of rope, or suspenders.

If the wound is so situated that a constrictor cannot be applied, the artery should be compressed proximal to the point of injury.

Venous hæmorrhage will be increased if the veins are constricted above the injury, but enough pressure is not exerted to completely close the artery. The femoral artery may be compressed against the pubic bone with the second and third fingers of the left hand, reinforced if need be by the right thumb. The subclavian artery in the supraclavicular fossa may be compressed against the first rib; the common carotid artery against the transverse processes of the cervical vertebræ. The entire fist may be used to compress the abdominal aorta against the lumbar vertebræ.

Hyperextension may be used instead of compression to control hæmorrhage from the femoral and subclavian arteries. The femoral artery may be stretched over and closed by the head of the femur if the thigh is hyperextended; the subclavian artery may be compressed between the clavicle and first rib if both elbows are bound behind the back, or if the arm on the side in question is drawn forcibly backward and to the opposite side.

Hæmorrhage is most dangerous from those arteries which, because of anatomical relations, are constricted or compressed with difficulty. In hæmorrhage from one of these vessels, compression should be resorted to immediately, the possibilities of infection being disregarded—as, for example, in hæmorrhage from the innominate artery or vein.

Permanent control of hæmorrhage is accomplished by ligation or suture of the injured vessel.

Ligation of Blood Vessels.—The ligature may be employed whenever closure of the vessel in question is not followed by severe nutritional disturbances. In ligating a vessel the injured point is first exposed under artificial ischæmia. If the vessel is completely divided, both ends should be seized with artery forceps, drawn somewhat out of the wound and ligated. In a lateral injury the vessel should be freed above and below the point of injury from its sheath, and then with the aid of an aneurysm needle a catgut ligature should be passed about the vessel above and below the wound. The segment of the vessel lying between the ligatures should then be resected.

Suture of Blood Vessels.—An attempt should be made to suture the vessels or vessel, if both the artery and vein are injured, or if ligation of the vessel is followed by severe nutritional disturbances, such as

occur frequently after ligation of the common carotid artery, more rarely after ligation of the femoral artery. A lateral or a circular suture may be performed, depending upon the conditions found at the time of operation.

Lateral suture was attempted before circular suture. The suture was first attempted upon veins, after it had been demonstrated by experimental work (Braun) and by clinical experience (Schede, 1882) that thrombosis did not occur if the operation was performed aseptically.

Important animal experiments in circular suture of vessels were performed by Gluck (1882) and by Jassinowsky (1891). The latter especially demonstrated that in spite of the technical difficulties, a circular suture can be inserted without secondary hæmorrhage or thrombus formation occurring and without an aneurysm developing. The first circular suture of an artery in man was performed by Murphy in 1897, the lumen of the femoral artery being reëstablished.

While the suture is being inserted the artery should be closed above and below the point of injury by digital pressure or by a delicate clamp (the blades of which are covered with rubber tubing, such as a Crile clamp). The ordinary hæmostatic forceps should not be used, as it injures the endothelium, causing thrombus formation.

[Carrel's method of suture is the most successful; a very fine needle and silk which is vaselined are used in performing the suture.]

A lateral suture may be employed in closing longitudinal wounds, and transverse and oblique wounds which do not involve more than one half of the circumference of the vessel. In applying this suture a fine non-cutting needle armed with the finest silk, which should be vaselined, is carried through all of the coats of the vessel, a continuous suture being inserted (Doerfler). The edges of the wound should be held firmly together, and the margin of the endothelium, in which the proliferative changes first occur, should be accurately approximated. Some interrupted sutures which include only the adventitia, and the connective tissues about the vessel should then be inserted to protect the line of suture. After the operation is completed, the forceps should be gradually removed so that the stitches may gradually tighten as the blood courses through the vessel. It is sometimes advisable to exert gentle pressure over the line of suture for a little while before closing the wound.

The same precautions should be taken in applying a circular suture as described above. The circular suture is employed to reunite the ends of a completely divided artery or close a large defect. It is more difficult to apply a circular than a lateral suture. Murphy's method, in which the proximal end is invaginated into the distal by sutures passed

through all the coats of the vessels, presents many technical difficulties. [Carrel's method is best suited for circular suture.]

Mechanical Methods for Repairing Vessels.—Of the different mechanical methods that introduced by Payr is the most successful. The magnesium tube used by him in making an anastomosis is very thin, measures from 0.3–1 cm. in length, and presents two grooves upon its outer surface. The tubes, of course, come in different sizes, which correspond to those of the arteries for which they are employed. In making an anastomosis, the proximal end of the artery or vein is drawn through the tube and is then everted by sutures or forceps so that the endothelium faces outward. The everted end is then tied with a fine silk ligature in the second groove. The tube covered by the proximal end is then slipped into the distal end, which is then tied over the tube. Endothelium is then applied to endothelium, and there is no foreign body in the blood stream. Union occurs within ten days, and then the magnesium tube is gradually absorbed. A distance of 5 cm. may be overcome by placing the extremity in the proper position, rendering suture without tension possible.

Naturally lateral and circular suture can only be performed on the larger vessels. Smaller vessels, about 3 mm. in diameter, are sutured with difficulty, and besides thrombosis is apt to occur.

Repair of Wounds in Vessels.—Agglutination of the margins of the wound, assisted by the formation of a thrombus composed of blood platelets and of a layer of fibrin, is the first step in the repair of arteries and veins after ligation or suture. Evidences of endothelial proliferation may be found a few hours after ligation or suture. The rapidly growing endothelial cells repair the defect upon the inner surface of the vessel, grow in between the edges of the wound, and cover the sutures which have been inserted. The fibrous elements of the media and adventitia soon proliferate and aid in repair. But few elastic fibers are found in the adventitia and media. They are, however, relatively numerous in the intima (Jacobsthal). The same changes are observed in the spontaneous healing of small wounds of vessels, provided the hæmorrhage is controlled by the resistance offered by the soft tissues. The clot which then closes the wound in the vessel wall is organized to form a scar.

VII. INJURIES OF LYMPHATIC VESSELS

Injuries of lymphatic vessels are of less importance surgically than are injuries of blood vessels.

Injury of the thoracic duct or of one of its largest branches is the most important of the injuries affecting lymphatics. The thoracic duct may be injured at its point of junction with the left subclavian vein

during the removal of deeply situated and adherent tuberculous lymph nodes. It may also be divided in stab or gunshot wounds of the supraclavicular fossa. The loss of chyle following its division may cause severe nutritional disturbance. Usually, however, the discharge of chyle gradually ceases as healing progresses. If the divided duct is seen in the wound, the thoracic end should be grasped by artery forceps and ligated. If the divided duct cannot be found, the external discharge of chyle can easily be prevented by packing the wound with gauze. In the majority of cases division of the duct is not followed by bad results, as a rich collateral circulation is soon established or the duct empties into the vein by a number of branches (Wendel).

Injuries of the thoracic portion of the duct may be associated with contusions of the chest and fractures of the thoracic vertebræ. If death is not caused by the injury to the chest or vertebræ, chyle is then poured out into both pleural cavities. Chylothorax may also be caused by the pressure exerted by tuberculous or carcinomatous lymph nodes upon the duct.

A chylous ascites may be caused in the same ways as mentioned above.

Exploratory puncture reveals a milky fluid which contains fat, albumin, and usually sugar, if there is a chylothorax or a chylous ascites.

Recovery may take place spontaneously after traumatic rupture of the duct, as the opening in it may be closed by the pressure of the extravasated fluid. If in chylothorax the respirations become embarrassed, the fluid should be aspirated. Only enough should be removed to relieve the respirations, as more fluid is poured out after aspiration.

Lymphatic vessels of different sizes are injured in all wounds and in all subcutaneous injuries. Large amounts of lymph, however, rarely accumulate, as the collateral lymphatic circulation is very free, and the lymph passes into other vessels. In many subcutaneous injuries lymph is extravasated and assists in the formation of the swelling. In subcutaneous separation of the skin (décollement) lymph mingles with the blood, giving rise to a characteristic clinical picture.

Dilated lymphatic vessels in the skin may rupture spontaneously or burst as the result of a blow or pressure. A permanent lymph fistula may then form.

LITERATURE.—*Apollonio.* Mikroskopische Untersuchungen über die Organisation des Unterbindungsthrombus in den Arterien. Beitr. z. pathol. Anat., Bd. 3, 1888.—*Baumgarten.* Ueber die Schicksale des Blutes in doppelt unterbundenen Gefässtrecken. Wien. med. Wochenschr., 1902, No. 45;—Ueber die sog. Organisation des Thrombus. Centralbl. f. die med. Wissensch., 1876, p. 593.—*v. Brunn.* Beitrag zur traumatischen Gangrän durch Ruptur der inneren Arterienhäute. Beitr. z. klin. Chir., Bd. 41, 1903.—*Delbet.* Maladies chirurg. des Artères, Dentu et Delbet. Traité de chir., Paris, 1897, p. 141, Part IV.—*Dörfler.* Ueber Arteriennaht. Beitr. z. klin.

Chir., Bd. 25, 1899, p. 781.—*Fischer.* Ueber die Gefahren des Lufteintrittes in die Venen während einer Operation. v. Volkmanns Samml. klin. Vorträge, 1877, No. 113.—*Hare.* The Entrance of Air into the Veins. Americ. Journal of the Med. Sciences, 1902, November.—*Heller.* Ueber traumatische Pfortaderthrombose. Verhandl. d. path. Gesellsch., Zentralbl. f. allgem. Pathol., Bd. 15, Ergänzungsheft, p. 182, 1904.—*Höpfner.* Ueber Gefässnaht, Gefässtransplantationen und Replantation von amputierten Extremitäten. Arbeiten aus v. Bergmanns Klinik, 17, 1904, mit Lit. und Arch. f. klin. Chir., Bd. 70, 1903, p. 417.—*Jacobsthal.* Zur Histologie der Arteriennaht. Beitr. z. klin. Chir., Bd. 27, 1900, p. 199.—*Jordan.* Luftaspiration in die Venen des Halses. Handb. d. prakt. Chir., 2. Aufl., Bd. 2, p. 43.—*Körte.* Ueber Gefässverletzungen bei Verrenkungen des Oberarmes. Arch. f. klin. Chir., Bd. 27, 1882, p. 631.—*Kümmell.* Chylothorax. Mit Lit. im Handb. d. prakt. Chir., 2. Aufl., Bd. 2, p. 497.—*Linser.* Ueber Zirkulationsstörungen im Gehirn nach Unterbindung der Vena jugul. int. Beitr. z. klin. Chir., Bd. 28, 1900, p. 642.—*Marchand.* Der Prozess der Wundheilung. Deutsche Chir., 1901, Wunden der Gefässe, p. 330.—*Payr.* Beiträge zur Technik der Blutgefäss- und Nervennaht. Chir. Kongr.-Verhandl., 1900, II, p. 593 and Arch. f. klin. Chir., Bd. 62, p. 67;—Weitere Mitteilungen über Verwendung des Magnesium bei der Naht der Blutgefässe. Ibid., Bd. 64, 1901;—Zur Frage der zirkulären Vereinigung von Blutgefässen mit resorbierbaren Prothesen. Ibid., Bd. 72, 1903.—*E. Pick.* Ueber die Rolle der Endothelien bei der Endarteritis post ligaturam. Zeitschr. f. Heilkunde, Bd. 6, 1885, p. 457.—*Raab.* Ueber die Entwicklung der Narbe im Blutgefäss nach der Unterbindung. Arch. f. klin. Chir., Bd. 23, 1879, p. 156;—Neue Beiträge zur Kenntnis der anatomischen Vorgänge nach Unterbindung der Blutgefässe beim Menschen. Virchows Arch., Bd. 75, 1879, p. 451.—*Rotter.* Ueber Stichverletzungen der Schlüsselbeingefässe. v. Volkmanns Samml. klin. Vortr., 1893, N. F., No. 72.—*Schmitz.* Die Arteriennaht. Deutsche Zeitschr. f. Chir., Bd. 66, 1903.—*Schopf.* Verletzungen des Halsteiles des Ductus thoracicus. Wien. klin. Wochenschrift, 1901, No. 48.—*Senn.* An Experiment and Clinical Study of Air-Embolism. Centralbl. f. Chir., 1886, No. 23.—*Thöle.* Querdurchtrennung des Duct. thoracicus am Halse. Deutsche Zeitschr. f. Chir., Bd. 58, 1900, p. 95.—*v. Wahl.* Die Diagnose der Arterienverletzungen. v. Volkmanns Samml. klin. Vortr., 1885, No. 258.—*Wendel.* Ueber die Verletzung des Ductus thoracicus am Halse und ihre Heilungsmöglichkeit. Deutsche Zeitschr. f. Chir., Bd. 48, 1898, p. 437.

VIII. INJURIES OF JOINTS

(a) SUBCUTANEOUS INJURIES

Subcutaneous injuries of joints may be divided into contusions, sprains, and dislocations.

A contusion may be caused by direct or indirect violence. In the former the force is applied over the joint, while in the latter it is transmitted from some distant point, the articular surfaces being driven together. For example, the knee or hip joints may be contused by a fall upon the feet, the elbow joint by a fall upon the hand.

In contusions caused by direct force the soft tissues surrounding the joint are injured as well as the synovial membrane. In contusions caused by indirect force, fragments of bone may be separated, the articular surfaces fissured, the spongy ends of the bones crushed, and the articular

cartilage separated from the subjacent bone by an accumulation of blood.

Hæmarthrosis.—The most prominent symptom of a contusion of a joint is an extravasation of blood into the joint cavity (hæmarthrosis). The contour of the joint is changed, as its normal lines are obliterated by the distention of the capsule. The hæmarthrosis develops rapidly after the injury, and reaches its maximum development on the following day. Movements of the joint involved are painful and its function is interfered with.

The prognosis of a contusion uncomplicated by a fracture and associated with but a small extravasation of blood is good. Recovery with good function usually occurs within a short while.

Functional disturbance may develop if the blood is incompletely or slowly absorbed. The absorbing power of synovial membrane is not great, for the lymphatic plexuses are not in direct communication with the joint cavity, differing in this way from the lymphatic plexuses of serous cavities. Large exudates are not removed unless absorption is favored by compression, massage, and gentle active and passive movements, or unless the capsule is torn opening in this way lymphatics of the surrounding tissues. Coagulated blood causes the greatest disturbance.

Riedel has shown by experimental work on animals that one third of the blood poured out into a joint becomes coagulated. The changes which blood undergoes in a human joint vary. Sometimes coagulation occurs early, while in other cases the blood remains fluid for a number of weeks. In those cases in which the blood remains fluid for a long time there is always considerable fat in the joint cavity which is apparently derived from contused bone marrow. Fat probably prevents or delays the coagulation of blood.

Regressive changes occur much more slowly in clotted than in fluid blood, and clotted blood is absorbed much less rapidly. The irritation of the synovial membrane induced by the blood may be followed by a serous exudate, giving rise to the clinical picture of a chronic or recurring hydrops. Long-continued immobilization of such a joint may be followed by organization of the fibrinous masses lying between the articular surfaces, causing fibrous anchylosis. A large chronic exudate may so distend and weaken the capsule that the joint becomes flail. Other complications of a contusion are subcutaneous suppuration and tuberculosis of the injured joint. They may develop as hæmatogenous infections or after the rupture by the force causing the contusion of the capsule of some latent focus. Infections of the hæmatoma and joint may develop from small wounds in the skin and from excoriations.

Diagnosis.—The diagnosis of a simple contusion of a joint, uncomplicated by a fracture, is based upon the character of the injury and the findings elicited by an examination. Distention of the capsule, fluctuation, the so-called "snowball" crepitation, which can be elicited by pressure at certain points and is caused by displacement of blood clots, and the signs of a contusion of the soft tissues are indicative of a hæmarthrosis. Severe pain elicited by pressure made at definite points and abnormal lateral mobility indicate the laceration of ligaments. Fractures involving most of the joints are very typical. Fissures and fractures of the epiphysis without displacement cannot be recognized unless a Roentgen-ray examination is made.

Treatment.—The first indication in the treatment of a contusion of a joint is to put the part at rest. If one of the joints of the lower extremity is involved the patient should remain in bed. If a bandage which exercises mild compression is applied immediately, and the joint is then immobilized upon a papier-maché or a molded plaster-of-Paris splint, the pain is relieved more rapidly and less blood is poured out into the joint than when an icebag is applied and massage is employed to hasten absorption. The immobilizing dressing should not be employed longer than one week, provided there is no fracture. At the end of a week massage, active and passive motion, and baths should be begun in order to prevent stiffness of the joint. No weight, however, should be borne upon the joint at this time. If the blood is absorbed slowly and the exudate is large, it may be necessary and advantageous to aspirate the joint. After aspiration a dressing which exercises mild compression should be applied over the joint. Such a dressing should be worn for some weeks.

Sprains.—An injury in which there is a sudden momentary displacement of bones entering into a joint, the parts returning immediately to their normal relations, is classified as a sprain.

A sprain may be caused by movements carried beyond the normal range of motion peculiar to the joint (for example, a sprain caused by hyperflexion or hyperextension of a hinge joint), or by some movement which normally does not occur in the joint involved (for example, a sprain of a hinge-joint caused by forceful attempts at rotation). The cause of sprains of the various joints differs. The most frequent and best-known examples are sprains of the ankle caused by a misstep, and sprains of the wrist caused by falling upon the flexed or extended hand. Next in order of frequency are sprains of the knee caused by abduction or rotation of the leg. Joints with a very free range of motion, such as the shoulder and hip, are more frequently dislocated than sprained.

Pathology of a Sprain.—The capsule and ligaments are nearly always lacerated at the points where they have been exposed to the greatest

tension during the exaggerated or abnormal movements. The tear may involve one or all of the layers of the capsule and may vary greatly in extent. Strong accessory bands in a capsular ligament are often very resistant and frequently are not torn, a piece of bone to which they are attached being torn away (as in Colles' and Pott's fractures). Bony prominences, such as the coracoid process, tendons and muscles, intimately related to the joint and inhibiting the movements of the same, may be torn off and partially or completely ruptured. Intra-articular fibrocartilages may be displaced and parts of the capsule or tendons and muscle may become caught between the articular surfaces.

Symptoms.—The first symptom of a sprain is severe pain. The pain soon subsides if only the capsular ligament is lacerated. If the ligaments are badly torn or if there is also a fracture, the pain persists until the part is immobilized. Within a few hours the joint becomes swollen, as blood is extravasated into the joint cavity and periarticular tissues. Ecchymoses develop in the skin surrounding the joint, especially at the points where the ligaments have been lacerated or a fragment of bone separated. Movements are avoided and no attempt is made to bear weight upon the joint, as the pain is increased thereby.

Clinical Course and Prognosis.—The clinical course and prognosis of a sprain are much the same as those of a contusion. Simple sprains without extensive laceration of the ligaments or injuries of bones go on to complete recovery in a short time. If, on the other hand, the articular cartilages are displaced or a piece of the capsule becomes incarcerated, the pain persists for a long while. A fracture, if not recognized or if neglected, may heal in malposition, causing marked functional disturbance. Too early use of an extremity after the laceration of important ligaments may cause abnormal mobility (lateral mobility of the knee joint), and faulty positions (genu valgum, pes valgus). Sprains readily recur, as the ligaments which may not have completely healed become weak and relaxed when used early.

Diagnosis.—The diagnosis of a sprain is not always easily made. The character of the injury, and the development of a painful swelling of the joint which interferes with every motion make the diagnosis of a sprain probable. The laceration of a ligament, if there is no fracture, is recognized by tender points corresponding to the position of the tear. In sprains of the ankle joint these tender points are found over the deltoid ligament; in sprains of the knee joint about the joint line. Even if but a small hæmatoma has developed, these points will be more resistant than corresponding points upon the uninjured side.

The deformities associated with fractures involving joints (Pott's and Colles' fractures, fracture of the patella) are usually characteristic

and typical. These fractures should not be mistaken for sprains. Quite frequently, however, but a small fragment of bone is broken off and the typical deformity is concealed by a hæmatoma. In doubtful cases a positive diagnosis can be made by a Roentgen-ray examination. Roentgen-ray examinations have shown that small fragments of bone are more frequently torn off in sprains, and that fissures are more common than clinical findings would seem to indicate. The presence or absence of injuries to muscles and tendons should be determined by palpation and a test of function.

Treatment.—The treatment should be the same as already described in discussing contusions of joints. Only when the exudate is small, when the interference with function is not marked, and there is no laceration of the ligaments should massage be begun immediately. In all other cases an immobilizing dressing should be employed for at least one week. If the ligaments are extensively lacerated, or if there is a fracture, an immobilizing dressing should be worn for a number of weeks. It should be so applied, however, that it can be easily removed, so that massage may be employed and gentle active and passive motion begun when indicated.

Persistent, marked functional disturbances, which may be caused by incarceration of a part of the capsule, or extensive laceration of the ligaments or displacement of the semilunar cartilages, may demand operative interference. An incarcerated portion of the capsule should be removed, a lacerated ligament sutured, a displaced semilunar cartilage sutured in place or removed.

The tissues along the lines of attachment of the capsular and intracapsular ligaments may be crushed or lacerated in both severe and insignificant contusions and sprains. Such injuries are especially apt to occur at the points of attachment of the crucial ligaments of the knee joint. Small fragments of cartilage may be torn off when considerable tension is exerted upon the ligaments. The separation of small pieces of cartilage, together with small fragments of bone, occurs most frequently in the knee and elbow joints.

Floating Cartilages.—These fragments, which may become quite large, measuring several centimeters in diameter, may be partially or completely separated from the articular cartilages. They form one class of the so-called joint bodies or "*joint mice.*" Apparently the separation from the articular cartilage takes place slowly, and is due to the granulation tissue which develops after a piece of cartilage has been partially detached by some trauma, or has become necrotic as the result of an injury (König). A history of a previous injury to the joint can usually be elicited in these cases of floating cartilage. Then after a period varying in length from days to years the characteristic symptoms

of a floating cartilage—*locking of the joint and subsequent synovitis*—develop. The time intervening between the injury and the development of the symptoms of a floating cartilage depends upon the length of time required for the separation of the partially detached or necrotic piece of cartilage. The symptoms caused by traumatic floating cartilages do not differ from those caused by pathological floating cartilages (*vide* Arthritis Deformans). The symptoms associated with the former recur more frequently. Incarceration of a floating cartilage between the articular surfaces of a joint produces severe pain and inhibits motion. The pain persists and the joint remains locked until the cartilage escapes from between the articular surfaces spontaneously, or as the result of appropriate manipulations which the individual soon learns to make after the joint has been locked a few times. A serous synovitis develops after the joint has been unlocked. If the joint becomes locked repeatedly the serous synovitis becomes chronic and the capsule becomes thickened.

Some traumatic floating cartilages are absorbed, while others remain and are altered, as has been determined by animal experiments and histological examinations (Hildebrand, Barth, A. Schmitt, Schmieden, and others). A floating cartilage may become infiltrated and digested by the granulation tissue which develops from the synovial membrane or articular cartilage. Frequently, according to Barth, the absorption is not complete, the rough surfaces of the cartilage being merely smoothed off. The floating cartilage may then remain attached to the synovial membrane, later being surrounded by osteoid and cartilaginous tissue. If the pedicle attaching it to the synovial membrane is ruptured by some slight trauma the body becomes free again. Separated fragments of cartilage which do not become attached to the synovial membrane may also be retained. Both regressive and progressive changes may occur in these fragments (Schmieden). A joint body may become calcified after persisting for some time.

The shape of a floating cartilage may be changed. A joint body which has but recently become detached may be recognized by the smooth layers of the articular cartilage and the irregular processes of spongy bone which lie beneath the latter. Later the joint body becomes completely covered by nodular elevations of cartilage, and the traumatic can no longer be differentiated from the pathological variety. Remnants of articular cartilage, which may be recognized with difficulty in old calcified preparations, may indicate the origin of these floating cartilages. Even if articular cartilage is found, it cannot be said that the joint body is of traumatic origin, for, according to König, pieces of articular cartilage become separated spontaneously in *osteochondritis dissecans.*

Dislocations.—A dislocation is a permanent, abnormal, total, or partial displacement from each other of the articular portions of the bones entering into the formation of a joint (Stimson). A partial or incomplete dislocation is also called a subluxation.

Dislocations are classified as traumatic, congenital, and pathological.

Traumatic dislocations may be caused by direct or indirect violence, the bones entering into the joint being separated by force, or as the result of movements carried beyond the normal range of motion of the joint involved. Dislocations of the shoulder, for example, are usually caused by indirect violence, the arm being hyperabducted. When the arm is hyperabducted the upper end of the humerus comes in contact with the edge of the acromion, and a new center of motion is created if hyperabduction is continued. If the long arm of the lever, represented by the part of the extremity distal to the head is still hyperabducted, the head of the humerus, corresponding to the short arm of the lever, is forced out of the lower, weak portion of the capsule. Dislocations are rarely caused by direct violence.

Dislocations are also rarely caused by muscular action. The shoulder is occasionally dislocated by throwing the arm suddenly backward or in lifting a heavy weight. Many individuals can voluntarily dislocate different joints, most commonly the first metacarpo-phalangeal joint. A joint may be dislocated in a convulsion—for example, in epilepsy.

A habitual dislocation is a special form which recurs frequently, and may be caused by some insignificant trauma, by voluntary or involuntary muscular action. This form of dislocation is usually the result of an ordinary traumatic dislocation in which the ligaments were severely lacerated, or of a paralysis of one or more of the muscles about the joint, or of a fracture of one of the bony prominences about the joint which normally inhibits excessive movements.

After the head of a bone has been forced out of the capsule by torsion, hyperflexion, or hyperextension, it may be still further displaced. For example, in dislocation of the shoulder the head of the humerus is driven out of the lower part of the capsule by hyperabduction, and is then displaced forward beneath the coracoid processes. The secondary displacements are caused by the continued action of the force causing the dislocation, by the weight of the extremity, by the elastic tension of the soft tissues, and especially by the ligaments and muscles.

The nomenclature of the different varieties of dislocation has not been definitely established. Usually, however, the bone situated farthest from the trunk or the one which moves most freely upon the other is spoken of as being dislocated.

In speaking of a dislocation of the shoulder, it is described as a dis-

location of the upper end of the humerus. The position which the bone occupies after the dislocation has occurred, or the direction in which the bone is displaced, is employed in describing a dislocation; for example, one speaks of a subcoracoid dislocation of the shoulder, of a backward dislocation of the ulna. This nomenclature, however, does not always accurately describe the mechanism; for example, in the so-called backward dislocations of the elbow the lower end of the humerus is forced forward through the rent in the capsule, in the so-called dorsal dislocation of the fingers the head of the metacarpal bone is forced forward through the capsule.

Pathology of Unreduced Dislocations.—The pain and swelling soon subside, even when the dislocation is not reduced. A limited amount of motion returns, and the atrophic muscles become stronger. Changes occur in the articular cartilages, which are very similar to those associated with arthritis deformans. The concave articular surfaces become filled in with granulation tissue. The dislocated end of the bone rests in a depression which develops as the result of pressure, and is surrounded by an acetabulumlike wall of bone which is formed by the traumatized periosteum.

The scar tissue which forms surrounds the head of the bone like a new capsule. If the new depression becomes lined with fibrous cartilage, a *nearthrosis* (which permits of some motion unassociated with pain) develops.

Symptoms.—The symptoms of a recent dislocation are severe pain and more or less complete impairment of function. To these may be added the signs associated with changes in the position of the articular ends of the bones. These differ greatly, but are quite characteristic and typical for the different joints.

Diagnosis.—In making a diagnosis of a dislocation the character of the trauma, the impairment of function, and the findings elicited by inspection, palpation, and the Roentgen-ray picture should be considered.

Inspection reveals at once a striking change in the contour of the joint and in the direction of the bones entering into the formation of the joint. The form of the joint differs markedly from that of the sound side. Where normally there should be a prominence will be found a depression; where there should be a depression will be found a prominence. The direction of the dislocated bone is also changed. The axis of the bone no longer passes into the articular cavity, but passes by it. Besides the extremity is either shortened or lengthened, depending upon whether the end of the bone rests above or below the rim of the joint cavity.

If there is but little swelling, the findings revealed by inspection may be characteristic enough to make the diagnosis of a dislocation positive.

If so, palpation, which is often painful, may be omitted. If the swelling is great, careful palpation of the bony prominences of the joint, comparing them with those of the sound side, is of great value. The dislocated end of the bone may be felt in its abnormal position when the shaft of the bone is moved, and a depression may be felt at the position normally occupied by the dislocated bone. Passive movements will show that the dislocated head is firmly held in its abnormal position by tension of the muscles and ligaments, and that if the end of the bone is displaced from its abnormal position it quickly returns to it when traction is no longer made.

Complications.—Fractures are the most frequent complications of dislocations. If the line of fracture is situated close to the head of the bone or passes through the diaphysis, the head will no longer rotate with the shaft and the dislocated part will not be as immobile as is the case when a dislocation is not complicated by a fracture. The separation of small fragments of bones to which muscles or ligaments are attached may often be recognized by crepitus elicited when movements are made. Clinically it is often impossible to recognize the separation of these small fragments. A diagnosis can, however, be easily made if a Roentgen-ray examination is made.

One of the bones of the forearm or leg may be dislocated, the other fractured. Injuries of nerves, blood vessels, muscles, and tendons are less common than fractures. They may be recognized by the clinical symptoms peculiar to them.

An old dislocation is less painful than a recent one. Both active and passive motions are greatly restricted in an old dislocation, and crepitus which is caused by a destruction of the articular cartilages is usually easily elicited. There may be considerable swelling of the paraarticular tissue if repeated unsuccessful attempts at reduction have been made.

General Rules for the Reduction of a Dislocation.—The treatment of a dislocation consists of reduction, by which is understood the replacement of the dislocated end of the bone to its normal position. Although the manipulations that must be made in reducing a dislocation vary with the different joints, there is a general principle which underlies the reduction of all dislocations. [Gunn, of Chicago, and Bigelow, of Boston, were the first to suggest, as the result of careful anatomical studies and clinical experience, that the untorn portion of the capsule offered the main obstacle to reduction. Gunn then enunciated the following rule, which may be employed in the reduction of any dislocation: Relax the untorn portion of the capsule by placing the part in the position it occupied when the bone was dislocated and reverse the force.]

A dislocation can be more easily reduced if a general anæsthetic is

given. The muscles of a well-developed individual contract so powerfully that it is impossible to make the necessary manipulations unless they are relaxed. Laughing gas is to be preferred in these cases.

The earlier reduction is attempted the easier it will be. The adhesions which form in old cases must first be broken up by forcible manipulations before a reduction can be made.

After reduction has been attempted the surgeon should determine whether the bone occupies its normal position or not. If the attempt at reduction has been successful, an immobilizing dressing should be worn from eight to ten days. After this time repair has advanced far enough to permit of massage and gentle active and passive motion. The movements which caused the dislocation should not be attempted for some time, for the capsule may be stretched and torn again.

The function of the joint is soon restored after early reduction of a simple dislocation. Functional disturbances may persist for a long time if fragments of bone have been separated, or if a large amount of blood which is absorbed slowly is poured out into the soft tissues.

Conditions Rendering Bloodless Reduction Difficult or Impossible.—Bloodless reduction may be very difficult or impossible if the rent in the capsule is small and the capsule surrounds tightly the dislocated bone, or if a tendon (in dorsal dislocations of the thumb) or a piece of the capsule with or without a fragment of bone (in dislocations of the humerus) becomes incarcerated between the dislocated bone and the one with which it articulates.

Reduction by the Open Method.—In these cases it may be necessary to expose the joint by an open operation in order to remove the obstacle to reduction. An operation must also be performed in old dislocations which can no longer be reduced by the ordinary manipulation. The capsule must then be opened, the scar tissue removed from the joint cavity, adhesions separated, and the dislocated bone returned to its normal position. The earlier a reduction by the open method is attempted in these cases the better the functional results will be.

Reduction of a Dislocation Complicated by a Fracture.—It may be very difficult to reduce a dislocation complicated by a fracture. A dislocation complicated by the separation of a small fragment of bone, a tubercle, or tuberosity can often be reduced relatively easily, provided the piece of bone does not become incarcerated. But if the fracture is situated near the dislocated end of the bone or involves the diaphysis, it is only rarely that the dislocation can be reduced by the ordinary manipulation and direct pressure over the dislocated end of the bone. Then reduction by the open method is indicated, the fracture being wired at the same time. In these cases it is poor surgery to postpone the attempts at reduction until the fracture has united, for then at-

tempts at reduction are often unsuccessful, the bone is frequently refractured, and the function of the joint is restored slowly, if at all. In old dislocations with poor functional results resection of the head of the bone may be indicated.

(b) OPEN INJURIES OF JOINTS

Any kind of a wound may involve the capsule of a joint and open the joint cavity. A number of different foreign bodies, such as bullets, fragments of shells, needles, nails, pieces of metal, wood, and stone, may penetrate a joint cavity.

The symptoms and clinical course of such an injury depend upon the size of the wound and whether or not infection develops. Infection is the greatest danger associated with wounds of this character.

The opening in the joint produced by foreign bodies, punctured and gunshot wounds is usually small, and the most important diagnostic sign of an injury of joint—the discharge of synovial fluid—is frequently wanting. This is due to the fact that the wound may be closed by a blood clot or by a change in the position of the different tissues when the limb is moved. The position of the wound in the skin, the character of the injury, or the development of an exudate in the joint may be the only clew to an injury of this kind. A probe should never be used to determine whether a wound extends into a joint cavity or not, for the dangers of introducing a secondary infection in this way are great.

The swelling of a joint which develops immediately after a wound, especially a gunshot wound, is due to hæmorrhage; occasionally to the introduction of air. The swelling which develops later is due to inflammation. If the latter is caused by pyogenic or putrefactive bacteria severe local and general symptoms develop.

Most of the small wounds involving joints heal without complications if, after the area surrounding the wound in the skin has been carefully sterilized, a dressing of iodoform or dry aseptic gauze is applied and the part is immobilized in a circular or molded plaster-of-Paris dressing. It was shown by von Bergmann in the Russo-Turkish war that a foreign body may become encapsulated in a joint without causing infection if this conservative treatment is followed, and no secondary infection is introduced by attempting to locate the bullet by probing or by the introduction of special instruments. Aspiration is indicated if an exudate accompanied by mild symptoms develops. If the exudate is purulent the joint should be opened and drained. If a virulent suppurative or putrefactive inflammation develops, the joint should be opened widely, or resected, or an amputation performed, depending upon indications.

Penetrating foreign bodies (such as a splinter of wood, nail, or needle) should be removed immediately if they have rough or rusty surfaces, for clinical experience has shown that such bodies, when allowed to remain, are often followed by infection. Foreign bodies which irritate the synovial membrane, causing serous synovitis, or give rise to the symptoms associated with a free body should be removed, even if the wound has repaired completely without infection. Their position should be determined before an operation is undertaken by palpation and Roentgen-ray examinations, then but a small incision of the capsule is required for removal of the body. An immobilizing dressing should be worn for two weeks after such an operation.

Broad gaping wounds of joints are easily recognized. They are most frequently associated with incised and contused wounds (such as are caused by knives, sabers, and scythes), and with crushing injuries and lacerated wounds, such as are caused by the explosion of a bomb, the explosion of a boiler, by machines, and by wild animals.

Of these, the clean-cut, incised wounds heal most readily. If they are seen early the wounds in the capsule and skin should be sutured. Old cases of this character should be treated by the open method, and when the conditions become favorable secondary suture of the wounds should be performed.

Compound Dislocations.—Contused and lacerated wounds of joints may be associated with a dislocation. Compound dislocations are usually the result of great violence, the dislocated end of the bone being driven through the capsule surrounding it and the soft tissues over it. In other cases the destruction of the capsule is so extensive that the bone is displaced by its own weight. Complicating injuries of all kinds of the bones, nerves, blood vessels, muscles, and tendons may be associated with compound dislocations and those produced by great violence. In some cases strips and shreds of skin form the only connection between the dislocated part and the body.

The first indications in the treatment of a compound dislocation are to prevent infection and to favor wound repair. After these indications have been met, the dislocation should be reduced. The wound should then be treated by the open method, and the part immobilized by a molded or fenestrated plaster-of-Paris dressing.

Favorable cases recover with but slight impairment of function. Severe infections which may demand incision or resection of the joint, or amputation of the extremity, not infrequently develop.

LITERATURE.—*Barth.* Zur Lehre von den freien Gelenkkörpern. Chir. Kongr.-Verhandl., 1896, I, p. 31;—Die Entstehung und das Wachstum her freien Gelenkkörper. Arch. f. klin. Chir., Bd. 56, 1898, p. 507.—*Boerner.* Klin. u. pathol.-anatom. Beiträge zur Lehre von den Gelenkmäusen. Deutsche Zeitschrift f. Chir., Bd.

70, 1903.—*H. Braun.* Untersuchungen über den Bau der Synovialmembranen und Gelenkknorpel, sowie über die Resorption flüssiger und fester Körper aus den Gelenkhöhlen. Deutsche Zeitschr. f. Chir., Bd. 39, 1894, p. 35.—*van Hassel.* Du traitement des traumatismes articulaires. Journal de chir. et ann. de la soc. belge de chir., 1901, No. 6.—*Hildebrand.* Experiment. Beitrag zur Lehre von den freien Gelenkkörpern. Deutsche Zeitschr. f. Chir., Bd. 42, p. 292, 1896.—*Jaffé.* Ueber die Veränderungen der Synovialmembran bei Berührung mit Blut. Arch. f. klin. Chir., Bd. 54, 1896.—*Krönlein.* Die Lehre von den Luxationen. Deutsche Chir., 1882.—*Pagenstecher.* Die isolierte Zerreissung der Kreuzbänder des Kniese. Deutsche med. Wochenschr., 1903, No. 47.—*Riedel.* Ueber das Verhalten von Blut, sowie von indifferenten und differenten Fremdkörpern in den Gelenken. Deutsche Zeitschr. f. Chir., Bd. 12, 1879, p. 447.—*Schlatter.* Meniskusluxationen. Beitr. z. klin. Chir., Bd. 41, 1904, p. 229.—*Schmieden.* Ein Beitrag zur Lehre von den Gelenkmäusen. Arch. f. klin. Chir., Bd. 62, 1901, und Arbeiten aus v. Bergmanns Klinik 15, p. 209.—*v. Volkmann.* Die Krankheiten der Bewegungsorgane in v. Pithas und Billroths Handb. d. Chir. Erlangen, 1872, Bd. 2, Abt. 2, p. 618.—*Vollbrecht.* Umschriebene Binnenverletzungen des Kniegelenkes. Beitr. z. klin. Chir., Bd. 21, 1898, p. 216.

IX. INJURIES OF THE OSSEOUS SYSTEM

(a) SUBCUTANEOUS INJURIES OF BONES AND CARTILAGE

The results of the action of direct and indirect violence upon the osseous system differ widely, depending upon the character and the degree of the trauma. Slight changes, such as hæmorrhage beneath the periosteum, into the bone marrow, or spongiosa, may occur alone, or accompany the severer forms of fractures.

Subperiosteal Hæmatoma.—A subperiosteal hæmatoma may follow contusions or lacerations of the periosteum produced by muscular action or the displacement of the soft tissues, as the blood vessels entering the cortex may be torn in these ways. A painful, fluctuating swelling varying in size is caused by the accumulation of blood between the bone and periosteum. The so-called *cephalhæmatoma*, occurring so frequently over the one or the other of the parietal bones of the newborn, follows laceration of the periosteal vessels produced by the displacement of the scalp and the bones of the skull during labor. Traumatic separation of the perichondrium of the cartilages of the ear gives rise to a cystic swelling known as an *othæmatoma*. The blood extravasated in these cases remains fluid, and frequently it is not absorbed for a long time. The loosened periosteum proliferates at its points of attachment to the bone to form a thin wall of bone which surrounds the fluid blood like a wall. Occasionally the proliferation is still more extensive, and an entire piece of loosened periosteum becomes transformed into a thin scale of bone. Irregularities in the surface of the bone resulting from a proliferation of the periosteum may persist after the absorption of small extravasations of blood. These are quite common upon the anterior surface of the tibia.

Compression secured by a bandage evenly and firmly applied immediately after the injury and aspiration of the fluid blood are important factors in the successful treatment of these hæmatomas.

Hæmorrhages into the Bone Marrow.—Punctate hæmorrhages into the bone marrow follow concussions and accompany all fractures involving the larger bones. It has been demonstrated by the experimental studies of suppurative osteomyelitis that such hæmorrhages favor the deposition of bacteria from the blood stream. Further than this these hæmorrhages have no surgical significance.

Hæmorrhages, even when slight, and crushing injuries of the lamellæ may be followed by changes which may alter the form of the bones involved, and thus be the source of serious trouble (for example, changes in the form of the neck of the femur and of the vertebræ). An osteitis ending in sclerotic changes or a rarefying osteitis ending in atrophy may follow the development of granulation tissue resulting from such injuries.

Simple Fractures

By the term fracture, in the surgical sense of the word, is meant the breaking of a bone or cartilage. A fracture may be complete or incomplete, depending upon whether the bone is divided into two or more distinct fragments or not. A complete fracture is one in which the bone is divided into two or more distinct fragments by a line of fracture crossing its long axis. Fissures, " green-stick " fractures or infractions, the separation of a splinter of bone, or of an apophysis are classified as incomplete fractures.

Traumatic are differentiated from *pathological fractures* in which the resistance of the bone has been reduced by some inflammatory process (osteomyelitis, tuberculosis, syphilis), by tumors (sarcoma, carcinoma, enchondroma, cysts), or by some general disease (rickets, osteomalacia, scurvy, atrophy associated with paralysis, tabes, syringomyelia, etc.).

Fissures.—Fissured fractures are characterized by a split or crack in the bone which does not entirely circumscribe the fragment and does not separate it from the remainder of the bone. Fissures penetrate the bone involved in different directions and to different depths without altering its outline, as its continuity is maintained. The periosteum may remain intact or be elevated by an extravasation of blood, or torn. Aside from the tenderness and swelling following the development of the hæmatoma, there are no special symptoms. The diagnosis cannot be made with certainty except when the bone is exposed to direct examination. The larger fissures can, however, be distinctly seen in Roentgen-ray pictures.

Infractions.—An infraction or "green-stick" fracture is characterized by the fracture involving a portion of the thickness of a long bone, combined with a bending of the bone at the seat of fracture. It occurs most frequently in the costal cartilages and in the soft, yielding bones of children, especially those suffering with rickets. In this form of fracture the periosteum may remain intact or be torn at the seat of fracture. The integrity of the cortical layer of bone opposite the fracture is maintained.

Complete Fractures.—Complete fractures are caused by violence which exceeds the limits of elasticity and resistance of the bones upon which it acts. They may be produced by the contraction of powerful muscles, by a pull upon ligaments attached to the bone, or by direct or indirect violence.

The fracture may occur at the point of application of the force, or the force may be transferred along an extremity which is held rigid by muscular contraction or because of anatomical relations, and a bone some distance from the point of application of the force may then be fractured. Fracture of the clavicle by a fall upon the extended hand is an example of a fracture by indirect violence.

In considering the mechanism of a fracture, it will be readily seen that the force producing the fracture must overcome the elasticity of the bone and its resistance to bending (bending fractures) and torsion (torsion fractures), and its resistance to pressure (compression fractures) and pull (tearing fractures).

The resistance which the different bones offer to violence varies. The different forms of fractures depend upon the direction of the line of fracture and the mechanism by which they are produced. Fractures are classified as transverse, oblique, splintered, spiral, V-, T-, or Y-shaped, dentate, longitudinal, and comminuted, depending upon the direction of the line or lines of fracture. Depending upon the seat of fracture, they are spoken of as fracture of the shaft of the bone, separation of the epiphysis, and as intra-articular or articular. Depending upon the mechanism by which they are produced, the following classification is made (von Bruns, Helferich):

Bending fractures of long bones may be produced by direct or indirect force. If the force acts at right angles to the long axis of the bone, the latter is bent and broken like a green or tough stick when bent over the knee. If the force acts upon the long axis of the bone, the natural curves of the bone (for example, of the neck of the femur) become considerably exaggerated. Bending fractures are often characterized by the separation of a wedge-shaped piece of bone, the base of which always lies upon the concave side of the fracture. If this wedge-shaped piece is not completely separated an oblique fracture, combined with a fissure

of one fragment, is produced. In transverse fractures by bending the fissures in the bone may correspond to the form of a wedge.

Fissures and "green-stick fissures" may also be produced by this same mechanism of excessive bending.

The *flexion fracture* is very closely related to the fracture by bending. In this form one end of the bone is forcibly moved while the other end is held rigid. Bones are broken by this mechanism with the osteoclast, the fracture being produced at the point of fixation of the bone. Fracture of the olecranon by overextension of the forearm is another example of a fracture of this kind.

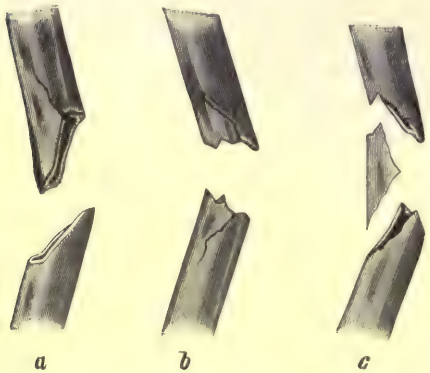


FIG. 224.—DIFFERENT FORMS OF BENDING FRACTURES. *a*, Oblique fracture; *b*, transverse fracture with fissures; *c*, oblique fracture with separation of a cuneiform fragment. (After Helferich.)

Compression or *crushing* fractures are produced in long bones by force acting in the long axis of the bone, the end of the diaphysis being driven into the broader and less resistant spongy mass of the metaphysis and epiphysis. Infractions then occur or the fractured ends become impacted. These fractures are most common in the neck of the femur following a fall upon the trochanter; in the upper end of the humerus and tibia following falls upon the elbow and foot respectively. Crushing fractures of the bodies of the vertebræ and of the os calcis may be produced by falls from a considerable height.

Fractures by crushing off occur when the projecting points of apposed articular surfaces are forcibly moved by each other. One of the best examples of this form is fracture of the coronoid process occurring in backward dislocations of both bones of the forearm.

Avulsion of an apophysis or a *scale of bone*, or a fracture involving one of the larger bones, may be produced by muscular action, or a severe pull upon a tendon or ligament attached to the bone. These fractures are frequently associated with dislocations. They occur most frequently in the typical Pott's and Colles's fractures, and in fractures of the olecranon and patella produced by muscular action.

Torsion fractures are produced by the forcible twisting of a bone when one of its extremities is held rigid. A bending of the bone is often a contributing factor in this form of fracture. The line of fracture runs in the form of a spiral, corresponding to the direction in which the bone has been twisted. The fragments of bone are pointed

and very easily penetrate the skin. Large screw-shaped fragments are frequently separated in this form of fractures (von Bruns, Fig. 225). The most common causes of torsion fractures are twisting of the body while the extremities are held fast, and injuries inflicted by the driving wheels of engines.

Comminuted fractures are produced by great violence. They occur most frequently in machine injuries, the bone being splintered into a number of fragments or crushed. They resemble closely fractures produced by projectiles.

FIG. 225.—SPIRAL FRACTURE OF THE FEMUR WITH A TYPICAL SCREW-SHAPED FRAGMENT. (von Bergmann's "Handbook of Practical Surgery.")

Injuries of the Soft Tissues, Viscera, and Nerves.—Injuries of the soft tissues, viscera, and nerves may be caused by the force producing the fracture or by the displacement of the bony fragments. Fractures by direct violence are accompanied by contusions of the skin and soft tissues, and for this reason larger hæmatomas develop in fractures caused by direct violence than in those caused by indirect violence. In fractures by indirect violence, especially in fractures by bending and torsion, the displaced fragments may injure the larger blood vessels and nerves, may penetrate and perforate the soft tissues, a simple fracture then becoming compound. In comminuted fractures the injury to the soft tissues may be very great, as the fragments may be forcibly driven into the surrounding tissues. The brain and spinal cord may be injured in fractures of the skull and vertebræ respectively; laceration of the urethra or bladder may be associated with fractures of the pelvic bones; contusion and laceration of the musculospiral nerve with fractures through the middle of the humerus.

Relative Frequency with which Different Bones are Fractured.—The statistics of von Bruns are of value in showing the relative frequency with which different bones are fractured. According to his statistics, fractures form more than one seventh of all injuries reporting for treatment. Bones of the extremities are more frequently fractured than are those of the head and trunk, fractures of the bones of the upper extremity being more common than those of the lower extremity. Fractures of the bones of the forearm are most common, forming 18 per cent of fractures; then follow those of the bones of the legs, ribs, and clavicle, forming from 15 to 16 per cent. Fractures of the bones of the hand form 11 per cent; of the humerus, 7 per cent; of the femur, 6 per cent; of the bones of the foot, 2.6 per cent; of the bones of the face, 2.4 per cent; of the skull, 1.4 per cent; of the scapula, vertebra, and pelvis, 1 per cent; and of the sternum, 0.1 per cent of all fractures. Fractures are four and a half times more common in the male than in the female sex.

Age at which Fractures are Most Common.—The greatest number of fractures occur in the third decennium. The frequency of fractures gradually increases from the tenth to the fortieth years, and then decreases. Still the frequency is greater in the eighth and ninth decennia than in the first and second. Fractures occurring at birth should, of course, be included in the first decennium.

Symptoms.—The symptoms of a simple fracture are subjective and objective. The former consist of pain at the point of fracture and impaired function, varying from slight to complete disability. The interference with function is due in part to pain which is intensified by movement, in part to loss of the support afforded when the bone or bones are intact. The most striking objective symptom is a deformity which is due to alteration in the shape of the bone and to displacement of the fragments. The deformity may be masked by an extravasation of blood, or may be entirely wanting if the fragments are not displaced. The second important objective symptom which can be determined in fractures which are complete and are not impacted is a *false point of motion.* When the fractured ends are manipulated, crepitus can be elicited at this point.

The character and the extent of the displacement of the fractured ends depend upon the continuance of the force producing the fracture, upon the weight of the part of the extremity distal to the fracture, upon muscular action, and upon the tension of the soft tissues. For example, in Colles's fracture, which is produced by a fall upon the palm of the hand, the lower fragment is displaced backward and upward by the force producing the fracture, while in fractures of the clavicle the acromial fragment is displaced downward and forward by the weight of the arm, and the clavicular fragment is drawn upward by the sterno-cleido-mastoid muscle.

The following table, taken from Stimson's "Fractures and Dislocations," is very valuable in showing the relative frequency of fractures of the different bones:

HUDSON STREET HOSPITAL, NEW YORK: STATISTICS OF FRACTURES TREATED IN HOSPITAL AND DISPENSARY, 1894–1903

	Cases		Cases	Per cent
Cranium	577	Head	577	4.78
Malar bone	16			
Nasal bones	571			
Superior maxilla	35	Face and Neck	1,063	8.65
Inferior maxilla	415			
Zygoma	23			
Hyoid	3			
Spine	62			
Pelvis	65			
Coccyx	3	Trunk	1,534	12.68
Sternum	8			
Ribs	1,396			
"Upper extremity"	132			
Clavicle	534			
Scapula	61			
Humerus, shaft and neck	325			
lower end	179			
internal epicondyle	6			
Radius and ulna	223	Upper extremity	5,781	47.89
Radius, shaft	316			
Colles's	1,007			
Ulna, shaft	272			
olecranon	102			
Carpus	14			
Metacarpus	873			
Phalanges	1,737			
Femur	422			
Patella	154			
Tibia, or tibia and fibula	764			
Abduction and adduction fractures at ankle	907			
Fibula	169			
External malleolus	27	Lower extremity	3,136	25.98
Internal malleolus	23			
Tarsus	136			
Metatarsus	255			
Toes	279			
Total	12,091			

Different Forms of Displacement.—Some forms of displacement are more common than others. Four principal forms are recognized:

1. *Angular displacement* is produced by the fracturing violence. Subsequently it is often increased by the weight of the part distal to

the seat of fracture, by contraction of the muscles when the patient attempts to move, and by other influences, such as spasmodic muscular contractions, when the parts are improperly immobilized. As a result of the action of one or more of the factors above mentioned an angular displacement may be transformed into one of the other varieties about to be mentioned.

2. *Lateral displacement* may take place forward, backward, or to either side, and may be partial or complete. The pure form of lateral displacement is rare and occurs only in transverse fractures. It is usually associated with overriding or angular displacement, or both.

3. *Displacement in the longitudinal axis* may be associated with shortening or lengthening of the extremity, depending upon whether the ends of the bones override or are separated by muscular action. In fractures through the diaphysis shortening is marked, especially in oblique and comminuted fractures, as the distal fragment is drawn upward by the muscles arising from bones above the seat of the fracture. Wide separation of the fragments may be produced by contraction of muscles attached to the proximal fragment. The most striking example of wide separation of fragments is seen in fractures of the olecranon process and patella.

4. *Rotatory displacements* usually occur in torsion fractures, and are caused by the fracturing violence or by some secondary factor, most frequently to the weight of the extremity distal to the line of fracture. In fractures of both bones of the leg the internal malleolus may be directed forward or even outward, while the patella occupies its normal position.

Displacement of the fractured ends in a transverse axis occurs in fractures of the head of the humerus and patella.

5. *Irregular displacements*, such as occur in comminuted fractures and in bursting and depressed fractures of the skull and facial bones, should also be mentioned.

Diagnosis.—In making a diagnosis of even a simple fracture, a definite line of procedure should be followed.

History of Accident, Pain, Impairment of Function.—The history of the accident and a description of the direction in which the force was applied or the blow delivered may give a clew to the diagnosis. Pain at the seat of fracture, and impairment of function, when attempts at movement are made, are suggestive but not conclusive, as similar symptoms are frequently associated with injuries of tendons and nerves. Whether the impairment of function is caused by a contusion, a sprain, a fissured fracture, or, if the loss of function is complete, by a dislocation must be determined by a more careful and exhaustive examination.

Inspection.—In making an examination the injured part should first be inspected and compared with the uninjured one, for frequently in typical fractures the findings revealed by inspection are so characteristic that there is but little need for manipulations which are often painful. A deformity, even when slight, will be apparent to the eye of a trained surgeon if the contour of the injured side is compared with that of the uninjured one. The deformity frequently becomes more pronounced when attempts at movement are made. If the injured part is greatly swollen and the deformity therefore masked, shortening of the extremity determined by use of the tape measure and the findings elicited by gentle manipulation are of great diagnostic value.

Palpation.—The diagnosis of a fracture can be made and the relative position of the fractured ends determined by palpation in most cases. If the finger is passed over the surface of a bone, a depression or fissure, or perhaps merely a loss of resistance, which indicates the position of the fracture, can be determined. Exquisite pain is usually elicited when pressure is made at the seat of fracture. This localized tenderness is an important diagnostic sign. If an articular end of a bone has been fractured and displaced, the end can usually be felt in a false position, and it can no longer be palpated in the position it ordinarily occupies.

False Point of Motion and Crepitus.—After inspection and palpation, an attempt may next be made to determine whether or not a false point of motion exists at the seat of deformity, or, if there is no deformity, at the point of tenderness. In eliciting a false point of motion, the part of the extremity distal to the fracture should be grasped and gentle traction made, while the proximal part is supported. The distal part is then either gently rotated or moved to and fro to determine whether or not a false point of motion exists. A false point of motion cannot be determined in impacted and green-stick fractures unless more force than is usually warrantable is employed. In determining whether or not there is a false point of motion in suspected fractures of the upper part of the femur and humerus, one hand should grasp the head of the bone while the other rotates the shaft. Naturally if there is a false point of motion the shaft and head of the bone will not rotate together. In fractures of the ribs and pelvic bones, the fingers should be placed upon either side of the supposed seat of fracture and alternately raised and depressed to determine whether a false point of motion exists or not.

A false point of motion is the most positive sign of a fracture. Crepitation should not be relied upon too much, as is so frequently the case with the beginner. In fractures involving the joints in which a false point of motion can only be determined with difficulty, if at all,

crepitation is of considerable value. When elicited in fractures of the shaft, it is a valuable diagnostic sign. It also indicates that the fractured ends are not overriding or separated by soft tissues. Crepitation elicited by pressure over the seat of the injury may be due to the displacement of blood clots, but crepitation elicited by torsion or movements is never caused in this way. The determination of a false point of motion and the elicitation of crepitus are, as a rule, painful to the patient and injurious to the soft tissues. They should be omitted whenever possible.

Roentgen-ray Examination.—The Roentgen-ray examination is the most important aid in the diagnosis of a fracture. It is essential that one should have an accurate knowledge of the shadows cast by normal bones taken at various angles in order that normal shadows and the lines representing epiphyseal cartilages will not be interpreted as fractures. When the dressings are changed a fluoroscopic examination should be made to determine whether or not the fragments are in apposition. If a deformity still persists, attempts at correction should be made.

In all cases a careful examination should be made to determine whether large blood vessels, nerves, and tendons have been contused and lacerated.

Traumatic and Pathological Fractures.—It may be exceedingly difficult to differentiate between a traumatic and a pathological fracture if the disease (tumor, chronic inflammation, atrophy) has given rise to no symptoms before the fracture occurred, or if trauma has been an accessory factor. If the trauma was not severe enough to have fractured a healthy bone, then the suspicion of abnormal fragility due to some pathological process should arise. If the Roentgen-ray picture gives no assistance in making the diagnosis, the subsequent clinical course alone can decide whether the fracture is pathological or not. Tumors which develop at the seat of a fracture are classified as " callus " tumors. They are usually sarcomas.

Clinical Course.—A swelling varying in size usually develops at the seat of fracture during the first two or three days. Discoloration of the skin which rapidly develops is due to the infiltration of the soft tissues with blood. It is most marked in fractures associated with displacement and comminution, for in these cases the soft tissues are contused and lacerated by the fracturing violence. Fractures involving the joints are always accompanied by an extravasation of blood into the joint cavity. Frequently the swelling is aggravated by an œdematous infiltration of the tissues, the result of an inflammatory reaction which, as a rule, continues but a few days. Quite frequently serous and serohæmorrhagic blebs develop in the swollen, tense, and discolored skin.

During the first week a slight elevation of temperature (so-called aseptic fever) is common. This fever is caused by the absorption of blood and tissue fluids from the tissues about the seat of fracture. Fat may be liberated and gain access to and circulate in the blood after crushing injuries of the bone marrow. If present in large amounts it may cause fat embolism, the symptoms of which vary depending upon the viscus or organ chiefly involved. The fat is excreted in the urine, and if there is a suspicion of fat embolism, which usually develops in from fifty-four to seventy-two hours, the urine should be carefully examined.

If the part is not immobilized soon after the fracture, the patient experiences severe pain, which is aggravated by manipulation and attempts at movement. The swelling and discomfort are usually greatly relieved by the application of a well-fitting, immobilizing dressing.

In from one to two weeks the swelling disappears and the pain subsides. Then a spindle-shaped swelling which surrounds the fractured ends can be felt. This swelling enlarges for several weeks, but gradually becomes smaller and harder, resulting in firm union of the fractured ends. This new tissue which develops at the seat of fracture is called the *callus.*

Callus Formation.—The regenerative changes leading to callus formation may be best followed in animal experiments. The first change indicative of regeneration consists of a proliferation of the cells of the periosteum and medulla. Proliferative changes may be seen within twenty-four hours after the fracture of a bone. A vascular granulation tissue develops from the periosteum, which frequently is lacerated and separated from the subjacent bone by blood clots. At the end of the first week islands of osteoid and cartilaginous tissue, between which lie marrow cells, are found within this newly formed tissue. Bone is formed from the external or periosteal callus, which extends some distance on either side of the fracture, as the result of the deposition of calcium salts. During this process the embryonal granulation tissue gradually becomes transformed into bone of an adult type. When the fractured ends are not properly immobilized and are subject to repeated displacements, the cartilage persists and is not transformed into bone. The internal or medullary callus forms more slowly in long bones than the external callus. It is composed of osteoblasts, which first produce osteoid tissue and later bone. The medullary cavity of the fractured ends of the bone are at first closed by this tissue.

If the fractured ends are held in apposition, bone formed by the periosteum and bone marrow rapidly unites them. If the ends are widely separated, the external and internal callus proliferate to fill in the gap and bridge over the space between them.

The fibrous tissue about a fracture assists in the formation of new

bone when its regenerative activity is stimulated by contusion and laceration. The proliferating fibrous tissue forms a granulation tissue which sends processes out into the intermuscular septa and bridges over joints, causing anchylosis. These changes resemble somewhat those observed in the traumatic forms of myositis ossificans. After fracture of adjacent bones union of the two masses of callus may lead to synostosis. Synostosis of the radius and ulna occasionally occurs after fracture of these two bones.

Consolidation of the Callus.—A callus gradually enlarges for four or five weeks and then undergoes ossification, which is, as a rule, completed in the following four weeks. The spongy mass of callus then becomes condensed and transformed into a less massive but firmer tissue, which resembles histologically the compact substance of normal bone. During this transformation the excessive callus is absorbed, the jagged ends of the bone are smoothed off, and the displaced splinters encapsulated or digested. The medullary canal is reëstablished when the fractured ends are in fairly good apposition in the same axis, but it remains closed when the ends override and are displaced longitudinally. After a few years a distinction can no longer be made between old and new bone, and only a small irregularity can be noted at the seat of the former fracture.

The Amount of Callus Formed.—The amount of callus formation in different fractures varies widely. A small amount of callus is formed in fractures with but little laceration of the periosteum and in fissures. An excessive amount of callus is formed when the periosteum is badly injured, when the bones are comminuted, and when the soft tissues are contused. Necrotic tissue and extravasated blood stimulate the tissues to proliferation and favor the development of large amounts of callus. Ordinarily callus formation is more marked in fractures of the diaphysis than in those of the epiphysis, but even in the latter, especially if comminuted, excessive callus formation is not at all infrequent. In fractures of the short and flat bones but little callus is formed, and it develops from the bone marrow.

Repair of Cartilage.—Fractures of the chondral and laryngeal cartilages are repaired by a callus which develops from fibrous tissues and later resembles histologically spongy bone. Fissures of the articular cartilages are repaired by fibrous tissue.

Time Required for Repair of a Fracture.—The time required for the repair of a simple fracture is usually about sixty days. Gurlt's statistics show that two weeks are required for the repair of fractures of the phalanges, three weeks for those of the metatarsal bones and ribs, four weeks for those of the clavicle, five weeks for those of the bones of the forearm, six weeks for those of the humerus and fibula,

seven weeks for those of the neck of the humerus and tibia, eight weeks for those of both bones of the leg, ten weeks for those of the shaft of the femur, twelve weeks for those of the neck of the femur. Consolidation occurs rapidly in children, and is complete in most bones in from two to three weeks. Union is much more rapid when the individual is healthy than when diseased.

Delayed and Non-union.—The causes of delayed and non-union may be local or general. If the general condition of the patient is bad as the result of previous sickness union may be delayed. Cachexia (following and associated with infectious diseases and malignant growths), senile marasmus, atrophy of bone associated with diseases of the central nervous system, and diseases of the bone, such as rickets and osteomalacia, interfere with callus formation. Among the local causes interfering with repair may be mentioned marked displacement and overriding of fragments, the interposition of soft tissues, large hæmatomas between the fractured ends, extensive destruction of the periosteum and medullary substance with comminution of the bone, poor nutrition of a fragment following a fracture into the joint, the separation of an apophysis or the thrombosis of the nutrient artery, and finally suppurative osteitis, the infection occurring at the time of the fracture or later through the blood.

Pseudarthrosis and Nearthrosis.—As a result of one of the above conditions callus formation may be either delayed or completely interfered with. If union is delayed, a marked enlargement persists for some time, but finally after a relatively long interval the callus becomes condensed and firm union occurs. Not infrequently when union is delayed the bone is refractured by attempts at movement, but the convalescence is not markedly prolonged, as the repeated insults stimulate the germinal tissue to the more rapid formation of solid bone. If bony union fails a false point of motion persists and a pseudarthrosis develops. In a pseudarthrosis the fractured ends are either separated by soft tissues or are united by a connective-tissue bridge. More rarely a true joint forms between the fragments, the ends of which are rounded off and covered with cartilage and enclosed in a mass of connective tissue resembling the capsule of a joint. Such a false joint contains a fluid resembling synovia, and simulates closely a joint which has undergone the pathological changes associated with arthritis deformans. The amount of interference with function in these cases depends upon the position of the bone involved. A pseudarthrosis involving a rib causes no functional disturbance, while a pseudarthrosis in one of the bones of the extremities prevents the bearing of weight upon the bone. At times the extremity may be used, after the fragments have been fixed by muscular contraction.

The bad results following simple fractures are usually due to associated injuries of the soft tissues, to the improper reduction and immobilization of the fragments, and to the loss of function following long-continued immobilization.

Complications.—Crushing of the bone marrow, which occurs to a greater or less extent in every fracture, may be followed by the absorption of fat and fat embolism. Injuries of arteries, veins, nerves, muscles, and tendons may be caused by the fracturing violence, or by the displacement of fragments insufficiently or improperly immobilized. Extravasations of blood, pulsating hæmatomas, traumatic aneurysms, thrombosis, gangrene of an extremity, paralysis, and muscular contractures may follow such injuries. Intra-articular fractures are accompanied by an extravasation of blood into the joint which frequently causes a synovitis and subsequent fibrous anchylosis. In intra-articular fractures the displaced fragment may interfere with motion, and besides free bodies may develop or changes resembling those of arthritis deformans occur after contusion and laceration of the tissues forming the joint. In some cases the changes following the extravasation of blood into the joint, such as serous synovitis and distention of the capsule, are very pronounced.

A number of functional disturbances which develop depend upon the conditions at the seat of the fracture. Besides functional disturbances caused by vicious union, pseudoarthrosis and synostosis, are those caused by the pressure of fragments of bone and callus upon the vessels and nerves. Thrombosis, interference with circulation, and nervous disturbances, which may even end in paralysis and are often caused by inclusion of nerves in the callus (the musculospiral nerve being frequently caught in the callus after fractures of the shaft of the humerus), are some of the sequelæ. Necrosis of the bone following infection and suppuration of the bone rarely occur in simple fractures.

Trophic Disturbances.—Long-continued disuse following immobilization of the injured extremity frequently leads to trophic disturbances. The beginning of trophic disturbances which usually develop after some weeks is indicated by a diminution in the size of the part. The skin becomes soft, thin, and smooth, the muscles atrophy as a result of disease and trophic changes in the spinal centers which follow peripheral irritation associated with the trauma or accompanying inflammation (Paget-Vulpian reflex atrophy). In these cases a Roentgen-ray picture taken after six or eight weeks reveals a marked atrophy of the bone and changes in its internal structure (Sudeck). The muscles and fascia atrophy, the tendons no longer glide freely in their sheaths, the function of the joints is interfered with, even when not injured, as the result of adhesions and degeneration of the articular cartilages. The

joint changes associated with immobilization are most marked in adults and old people. If a stiff joint is used or manipulated too roughly at first, an acute serous or serohæmorrhagic exudate may form as the result of laceration of the vessels in the contracted capsule. In children the growth of the entire extremity may be interfered with if a pseudarthrosis develops or the function of the part is interfered with as the result of anchylosis of one of the joints.

Œdema Following Fractures.—Circulatory disturbances are most common after fractures of the bones of the lower extremity. They appear after the first attempts are made to use the parts. Passive congestion and œdema are due to weakness of the muscles, which interferes with the venous circulation, and to venous thrombosis.

Decubitus and Hypostatic Pneumonia.—Old people are prone to develop bedsores and hypostatic pneumonia when confined to bed for any length of time.

Thrombosis and Embolism.—Pulmonary and cardiac embolism, which are usually fatal, may follow thrombosis of the veins about the fracture. A persistent and troublesome œdema, frequently associated with nutritional disturbances, is due to venous thrombosis. Thrombosis may be due to a weak action of the heart, to rupture and laceration of the deep veins, and to pressure exerted by the fractured ends of the bone or by the callus. These complications occur most frequently after fractures of the bones of the leg and in old people.

Persistent pain at the seat of fracture is another sequela which often interferes with the usefulness of the extremity. It is always present when the callus is recent, but usually disappears as it becomes older and more solid. In old people, however, it may persist or return after excessive use of the part or with every change of weather.

Prognosis.—The prognosis of fractures of the different bones differs widely. Fissures, green-stick fractures, and fractures with but slight displacement offer the best prognosis. The prognosis of comminuted fractures, of fractures with marked displacement, and of those associated with injuries of the soft parts and neighboring joints is bad, as shortening, pseudarthrosis, and stiffness may follow and persist. Fractures in young people heal much more readily than those in old people, and the prognosis is much better in healthy people than in those debilitated by chronic infections or constitutional diseases.

Statistics showing the results following different fractures are given by Haenel, Jottkowitz, Loew, Bliesener, Wolkowitch, and others.

Treatment.—The success of the treatment depends largely upon the way in which the first dressing is applied. In all fractures which occur outside of the patient's home, an emergency dressing should be applied which should immobilize the fragments during transportation to the

home or hospital, preventing the displacement of the fractured ends, so that they cannot injure the soft tissues, penetrate the skin, or cause excessive pain. A broken arm may be immobilized against the chest by a bandage or sling and supported by the uninjured arm. Temporary immobilization of fractures of the lower extremity is far more difficult. Wooden splints, broom handles, branches of trees, boards, and in war sabers and guns and other weapons, have been used to immobilize the parts during transportation of the wounded. These improvised splints are placed upon either side of the fractured part over the clothing, and extend far enough to immobilize the joints above and below the seat of fracture. In fractures of the femur the temporary immobilizing dressing should extend from the external aspect of the foot to the costal margin, and should be held in place by handkerchiefs, straps, cords, or suspenders. If there is nothing at hand which can be used to immobilize the part, the injured leg may be bound to the uninjured one. [A most useful dressing is the blanket splint, made by folding a blanket lengthwise once and rolling each end up into a firm roll over a lath or piece of wood. The fractured part is then placed between the rolls, which are held in place by three or four pieces of a roller bandage.]

If the individual is injured at home, he should be placed in bed and the fractured part should be immobilized between long sandbags. Frequently a physician is called to adjust a temporary dressing which will permit of easy and safe transportation of the patient to a hospital. In doing this the part should be handled gently and the following procedure should be followed: The parts above and below the seat of fracture should be held firmly, and the limb should then be gradually elevated, gentle traction being maintained upon the lower fragment. The clothing should then be removed. If there is any difficulty in doing this the clothes may be opened along the seam or cut off. The fracture should then be reduced as well as possible without anæsthesia, and after covering the bony prominence with cotton a wooden or a molded plaster-of-Paris splint should be applied.

The object of the treatment of a fracture is to secure firm union, in good position, of the fractured ends, and complete return of function as early as is compatible with the character of the fracture. Special works on fractures by Scudder, Hoffa, Helferich, and Stimson indicate the line of treatment which should be followed to secure good cosmetic and functional results in the different types of fractures.

Reduction of a Fracture.—The first indication in the treatment of a simple fracture is to bring the fractured ends in good apposition, thus correcting the deformity. This is called reduction or setting of a fracture.

When the displacement is great and severe pain is experienced when

the part is manipulated, the fracture should be reduced under gas anæsthesia. The skin about the part should be shaved and sterilized in order that the infection of hæmatomas and of the small cutaneous or serous blebs may be prevented.

Fractures of the bones of the trunk and face can usually be reduced by pressure. Many fractures into the joints may be reduced by changing the position of the joint involved. Reduction of fractures of the bones of the arm or leg should be performed as follows: The part proximal to the fracture should be grasped and held firmly by an assistant, and the surgeon should then grasp gently the distal fragment and exert traction. By traction, torsion, bending, or lateral motion combined with counterpressure, the deformity is reduced. If the proximal fragment is so short that it cannot be controlled, the distal fragment should be dressed in line with it. [A good example of this principle is afforded by fractures of the femur just below the lesser trochanter. The upper fragment is flexed, abducted, and rotated slightly outward by the action of the muscles attached to it. It is impossible to reduce and maintain this short fragment in position, so the long fragment, which can be controlled, is dressed in line with it. The fragment which can be controlled should always be dressed in line with the fragment which cannot be controlled.] At least one quarter of the fractured ends should be in apposition after reduction. The results of attempts at reduction can best be determined by Roentgen-ray examinations.

In fractures of the olecranon the forearm should be dressed in extension. In fractures of the upper third of the femur the lower fragment should be flexed, abducted, and rotated outward. Fractures about joints should always be dressed in the position of overcorrection; for example, in Colles's fracture the hand should be dressed in flexion, in Pott's fracture the foot should be rather strongly inverted.

Immobilization.—The fragments, when reduced, should be held in place by appropriate dressings. It is, as a rule, more difficult to maintain the fragments in good position than it is to reduce them.

Plaster-of-Paris Dressing.—If the injury is recent and there is no marked swelling of the soft parts, it is best to employ a plaster-of-Paris dressing, during the application of which the fragments should be maintained in correct position by traction and counterpressure. If there is considerable swelling, the extremity may be immobilized in a box or papier-maché splint until the swelling has subsided, and then a plaster-of-Paris dressing should be applied. The plaster-of-Paris bandage is employed in two ways: As a circular dressing, encasing the part (Matthysen, 1852), and as a molded splint (Beely, 1878). The bandage, as usually employed, consists of crinolin or cheese cloth, which is

infiltrated with powdered plaster of Paris. The bandage is applied after the extremity has been covered with a flannel bandage or cotton, which is held in position by a light mull or flannel bandage. Bony prominences and projecting tendons should be carefully protected. The bandage may be reinforced by pieces of wood, aluminum plates, or strips of tin.

Many considered the molded plaster-of-Paris dressing safer than the circular. In applying such a dressing to the leg, strips of flannel are cut of the desired length and width, and then a number of layers of a plaster bandage, enough to afford sufficient strength, are laid upon these. The dressing is then molded to the part and held in position by a roller bandage. This dressing can be easily removed when it is desired to massage the limb, and there is less danger of ischæmic contractures, gangrene, etc., when it is employed.

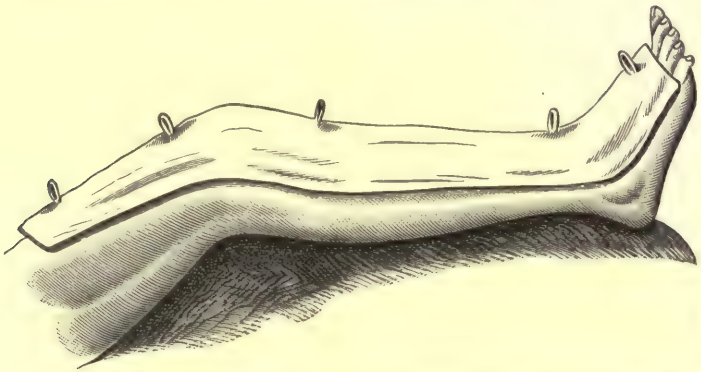


Fig. 226.—Beely's Molded Plaster-of-Paris Dressing for Fractures of the Leg. The dressing is provided with rings to be used for suspension.

Bad Results Following Poorly Applied Splints and Casts.—Any immobilizing dressing may be followed by bad results if applied too tightly or if the turns composing it are applied unevenly. When a cast is applied to an extremity, the fingers or toes, as the case may be, should not be included. They should be inspected frequently, their color and freedom of motion noted. The dressing should be split open or removed at once if the parts become congested or anæmic or paralyzed. The most serious consequences, such as gangrene of the extremity, thrombosis of the principal vessels, and ischæmic contractures, may follow the pressures of a poorly applied plaster-of-Paris dressing. Ulcers caused by pressure of a tight cast are a source of danger, as they provide infection atria for pyogenic bacteria. All bony prominences, sharp fragments, and prominent tendons should be padded in order to prevent pressure necrosis.

The first plaster-of-Paris dressing should be changed at the end of

the first week, or, at the latest, after the second week. The first dressing becomes loose when the swelling subsides, and, besides, the position of the fragments can often be improved and massage given when the dressing is changed. When the dressing is changed the skin should be washed off, and if there is an eczema it should be dressed with a dusting powder or an ointment. The immobilized joints and the muscles should be massaged before another dressing is applied, unless there is some contraindication.

Application of the Second Cast.—The amount of padding in the second cast may be greatly reduced as a rule. [Some advise applying the plaster-of-Paris dressing directly to the skin after it has been oiled or covered with vaseline. If a light flannel bandage is applied before the plaster dressing is put on, the patient will be more comfortable, and at the same time the parts will be better protected. There are no advantages in applying the cast directly upon oiled or vaselined skin.]

Ambulatory Dressing.—Convalescence is hastened if the muscles receive massage, atrophy being prevented in this way, and if the joints adjacent to the fracture are massaged and moved early. An ambulatory splint is employed in the treatment of fractures of the lower extremity by many surgeons. The best ambulatory splints are Hessing's molded iron splint, Thomas's hip splint, the molded iron splint of von Bruns, and the circular plaster casts advised by Krause, Bardeleben, Korsch, Albers, and others. When applied they are carefully molded to fit over bony prominences, such as the tibia, the condyles of the femur, the trochanter, and the pelvic bones in fractures of the femur. These dressings permit the patient to leave the bed before callus formation is complete, which is of the greatest importance in treating fractures in old people.

Extension Dressings.—The extension dressing devised by Buck is preferable in many cases to the iron splints and plaster-of-Paris dressing. This dressing is especially valuable in reducing and maintaining in correct position certain fractures characterized by marked displacement. In applying this dressing, broad strips of adhesive plaster are applied on either side of the limb and carried a little above the seat of fracture. These strips of adhesive plaster are attached to a foot- or hand-board, as the case may be, to which is attached a cord which runs over a pulley and is secured to a weight. The adhesive plaster is bound to the leg or arm by turns of a roller bandage. The cord usually supports a weight of from eight to ten pounds, and continuous longitudinal traction is made in this way. This dressing is effective in any position of the part. If the foot of the bed is elevated, the weight of the body usually affords sufficient counterextension. The extension dressing is frequently combined with Volkmann's sliding splint (Fig. 227). This

extension dressing has a number of advantages: (1) The displacement is gradually overcome without much pain, and the fragments are held in good position; (2) the part may be easily inspected and the position of the fragments improved, if necessary, by pressure and counterpressure; (3) the joints may be moved after a few days, and the stiffness

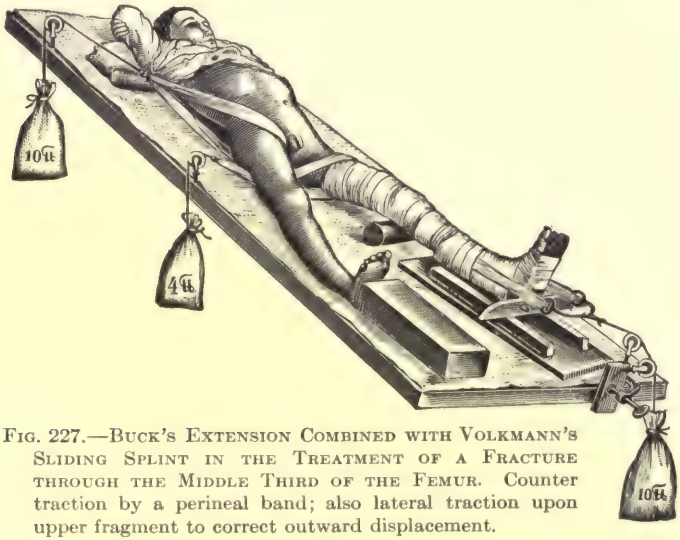


FIG. 227.—BUCK'S EXTENSION COMBINED WITH VOLKMANN'S SLIDING SPLINT IN THE TREATMENT OF A FRACTURE THROUGH THE MIDDLE THIRD OF THE FEMUR. Counter traction by a perineal band; also lateral traction upon upper fragment to correct outward displacement.

and synovitis associated with long-continued immobilization may be prevented (Bardenheuer). Repair of the fracture is not delayed, but rather hastened by the limited amount of motion permitted by such a dressing. Buck's extension has been generally adopted by the profession in the treatment of fractures of the shaft and neck of the femur. The same principle is being used more and more in the treatment of other fractures, especially those involving joints.

Bardenheuer has devised an adjustable apparatus for the treatment of fractures of the upper extremity, in which extension is secured by a spring, instead of by a weight and pulley.

Massage and early passive motion are important factors in securing a return of function. The function of the part may be impaired: (1) By stiffness of the joints and adhesions of the tendons to their sheaths; (2) by muscular atrophy; (3) by malposition of the fragments of the bone or bones.

Early Massage and Passive Motion in the Treatment of Fractures.—Stiffness of the joints and adhesions between the tendons and their sheaths may be the cause of severe functional disturbances. Lucas Championnière demonstrated some twenty-five years ago that in a num-

ber of fractures the fragments have no tendency to become displaced when they are once properly reduced, and that most excellent functional results could be obtained without any prolongation of the period of repair by early massage of the joints and tendons adjacent to the fracture. He also found that even in fractures with a tendency to marked displacement, the wearing of an immobilizing dressing for two weeks was sufficient. He has done a great service in emphasizing the so-called functional treatment of fractures, but has carried it too far.

The advantage to be derived from early motion and massage cannot be denied. An effort should, however, be made to maintain the fragments in good apposition, even if clinical experience has demonstrated that the functional results following union in poor position, but associated with free motion of the joints and the absence of muscular atrophy, are better than those following union of the fragments in perfect position, but associated with stiffness of the joints and atrophy of the muscles. Jordan and others pursue the proper course when they combine immobilization and the Championnière treatment. Bardenheuer lays great stress upon the treatment of fractures by extension combined with early motion and massage.

The fragments should be maintained in good apposition by immobilization or extension until union is firm enough to give fairly good support, but not as formerly, until the callus has undergone complete consolidation. In many fractures immobilization for two or three weeks is sufficient, but occasionally a much longer time is required. If the period of immobilization exceeds two weeks, massage should be employed, especially if the fracture involves a joint. An exception to this rule should be made in the treatment of fractures occurring in children. Early massage of a fracture in the young often leads to excessive callus formation, while even long-continued immobilization in a cast which may be required in difficult cases does not cause the slightest impairment of function.

FIG. 228.—SUPRACONDYLAR FRACTURE OF THE HUMERUS, USUAL TYPE. (von Bergmann's "Handbook of Practical Surgery.")

I do not believe that massage of a recent fracture is indicated. Immobilization continued for one week prevents pain and causes no impairment of function, even when the fracture involves a joint. Immo-

bilization protects against a number of complications, such as secondary hæmorrhage caused by displacement of the fragments and laceration of the soft tissues. The pain and swelling rapidly subside when an immobilizing dressing is properly applied.

Massage, when first applied, should consist of gentle rubbing of the seat of fracture, the part being rubbed from the periphery toward the trunk for fifteen or thirty minutes. If there are abrasions or large wounds of the skin, massage should not be begun until these are healed.

Osteoclasis and Reduction by the Open Method.—If union of the fragments has occurred in malposition it may be necessary to resort to osteoclasis. In cases in which it is impossible to reduce the fragments by manipulation and to maintain them in good apposition, it may be necessary to resort to reduction by the open method.

(*a*) *Osteoclasis*—artificial fracture of the callus—is resorted to in those cases in which union has occurred in malposition, as a result of neglect or imperfect immobilization, or because of the inherent difficulties of the case. If the callus is not consolidated it may be easily fractured by manual force and the fragments then placed in proper position. If consolidation has become complete, it may be necessary to use some form of an osteoclast. The osteoclast holds firmly the part of the bone above the fracture, and by slowly bending the distal segment the fracture can be produced at any desired point. In some apparatus (the Schneider-Mennel) the force is obtained by means of a pulley, traction being exerted upon the fragment to be broken.

(*b*) The *open method of reduction* has been employed for some time in the treatment of pseudarthrosis and in old cases which have healed in malposition. In this method the fracture is exposed, the connective tissue about the fracture removed, the bone is cut through by a chisel, and the fractured ends are given some definite form, so that they will fit together fairly well. The fragments are then maintained in apposition by sutures, ivory pegs, or nails. Frequently it is of advantage to chisel indentations or serrations in the fragments, so that they will fit together accurately.

The open method is frequently employed in the treatment of fractures when the fragments are widely separated by muscular action—for example, in the treatment of fractures of the olecranon, patella, and os calcis. A number of surgeons (Pfeil-Schneider, Lane, Tuffier, Fritz König, Martin, and others) employ the open method in the treatment of fractures in which the simple, closed method is employed by other surgeons.

Fritz König especially is a champion of the open method in the treatment of the various types of fractures. He considers it necessary

or at least of advantage in the treatment of fractures of the shafts of the femur and humerus with marked displacement or an interposition of soft tissues, of simple fractures of the shaft or ulna to avoid synostosis, of multiple fractures of the lower extremity, for the separation of fragments of bone caused by muscular action, for fractures involving joints with a rotation or dislocation of the fragment into the joint, for fractures associated with a dislocation, and for intracapsular fractures of the neck of the femur.

The most favorable time to attempt reduction by the open method is at the beginning of the second week (Fritz König). At this time the blood has been absorbed, the injured tissues are in a state of active regeneration, and the blood clot and the fragments of tissue which stimulate callus formation have become united with the surrounding parts. If an operation is performed early and the loosened tissues and the blood clots are removed, the bone is deprived of the natural stimulus supplied by them. A deficient callus which predisposes to pseudarthrosis is then apt to form.

(*c*) *Technic of Reduction by the Open Method, Bone Suture, etc.*— The fragments should be exposed under artificial ischæmia obtained by an Esmarch constrictor or a Martin bandage. After the ends of the fragments have been freed of fibrous tissue and approximated, they should be united by suture, wire nails, screws, ivory or bone pegs. In applying sutures the bones are first drilled and catgut, aluminum-bronze, or silver wire is passed through the drill holes and tied or twisted after the fragments are placed in apposition. Nickel-plated or silver-plated pins, bone or ivory pegs, silver-plated screws, and the bone plates introduced by Mr. Lane may also be used. If the wound can be closed, primary union usually occurs when the necessary aseptic precautions have been taken. Frequently deformities are not prevented, even when fractures of the diaphysis are reduced and fixed in the ordinary way by the open method. In these cases it is often advisable to use an intramedullary splint, a piece of bone or ivory peg being placed in the medullary canal and the fragments being approximated over it. [Murphy and Neff have obtained very good results in a number of cases by sawing off obliquely the ends of one of the fragments and then forcing this end into the medullary cavity of the other fragment. The results are the same as those obtained by use of a medullary splint, with this advantage that there is no foreign body to interfere with the process of repair.] When the medullary splint is used, a wire which includes the splint should be passed through the bone.

After closure of the wound an immobilizing dressing is applied as in simple reduction, and massage and passive motion are begun early.

Treatment of Delayed Union and Pseudarthrosis.—The treatment of delayed callus formation has already been discussed in the paragraph devoted to the bloodless treatment of pseudarthrosis.

Injections of alcohol, oil of turpentine, tincture of iodin, zinc chlorid, lactic acid, or blood freshly withdrawn from a vein are employed. Hot baths, massage, and rubbing together of the fragments may be used to stimulate and increase regeneration and callus formation. Passive hyperæmia obtained by applying daily an elastic constrictor proximal to the point of fracture for a period of from one to three hours may hasten repair.

Ambulatory splints may be of advantage in treating delayed union of the lower extremity, for the irritation to which the fragments are exposed when the patient is walking about hastens the development of bone. In many cases firm union which has been delayed for many months occurs after this treatment. It is a mistake to apply immobilizing dressings which cannot be easily removed and prevent early massage and passive motion.

Union following reduction by the open method is more rapid and complete when the freshened ends can be brought together directly. If a space remains between the fragments after dissecting out a nearthrosis or the fibrous tissue between them, there are a number of plastic procedures which may be resorted to. The defect may be overcome by transplanting bone taken from a neighboring bone; by resection of the neighboring bone, causing shortening of the part; by transplanting a section of dead bone which has been sterilized for some hours. The transplanted bone is attached to the fractured ends by sutures or is wedged into the medullary canal. In some cases it is advisable to take flaps of periosteum and bone from the fractured ends to bridge over the defect (W. Mueller, von Eiselsberg). In large defects of the tibia the method advised by Hahn, in which the lower fragment of the fibula is sawn through and then inserted into the medullary cavity of the tibia, may be used to advantage.

If, on account of the age, the general condition, or lowered resistance of the patient, a pseudarthrosis cannot be treated by the open method, a molded splint of the type suggested by Hessing should be employed. This splint is especially valuable in treating pseudarthrosis of the lower extremity.

(b) OPEN INJURIES OF BONES AND CARTILAGE

Different forms of wounds occur in bones and cartilage. Injuries of the periosteum and perichondrium are of less consequence than are injuries of bone and cartilage. Simple wounds of the cartilages of the nose, ears, larynx, and ribs are of no special significance, but open in-

juries (compound fractures) are always serious because of the dangers of infection, and associated injuries of important structures. Compound fractures associated with small, rapidly healing wounds may pursue much the same clinical course as simple fractures. Fractures associated with extensive laceration of the soft parts or almost complete separation of the extremity may be of secondary importance when compared to the gravity of the complications.

Relative Frequency of Compound Fractures.—The statistics of Gurlt, Weber, Moritz, and Billroth show that from 16 to 27 per cent of all fractures are compound. The following figures indicate the relative frequency of compound fractures in the different bones: 72 per cent of 88 fractures of the phalanges of the fingers and toes, 44 per cent of 52 fractures of the metacarpal and metatarsal bones were compound, while 17.9 per cent of the fractures of the leg, 11.6 per cent of those of the forearm, 7 per cent of those of the thigh, and 6 per cent of those of the humerus were compound (von Bruns).

A compound fracture may be produced by either direct or indirect violence, the object penetrating the soft tissues and fracturing the bone, or the fragments of the bone being forced through the soft tissue by the fracturing violence. A compound fracture by indirect violence may be caused by a displacement of the fragments at the time of the fracture, or from necrosis following pressure due to imperfect reduction of the deformity or careless treatment.

Decubitus developing over a sharp edge of a fragment may transform a simple into a compound fracture. Fractures of the nasal bones and of the base of the skull must usually be regarded as compound fractures, because the mucous membranes at the seat of the fracture are, as a rule, torn.

Fractures produced intentionally in operative work must be considered as a special class of compound fractures. A bone may be sawn or chiselled through in order to gain room for operative work (temporary resection of the mandible in removing a carcinoma of the tongue, of the clavicle, bones of the skull, ribs, etc.). Frequently long bones are divided obliquely or a cuneiform piece is removed to correct malformations due to bowing of the shaft or differences in length.

Fractures associated with gunshot wounds form another class of compound fractures. Complete separation of the extremities from the trunk, such as occasionally occurs in explosions, machinery, and railroad accidents, are closely related to compound fractures (Klauber).

The same principles should be followed in examining and making a diagnosis of a compound fracture as have been discussed in dealing

with simple fractures. It is a great mistake, often followed by secondary infection, to probe a wound associated with a fracture in order to determine whether it is superficial or extends to the point of fracture.

Treatment.—In serious cases the treatment should first be directed to counteracting the shock, which frequently is present in compound fractures, to controlling the hæmorrhage, and to preventing infection. Von Volkmann maintained that the first dressing determined the fate of the patient and the course of repair. His method (which consisted of opening the wound widely and sterilizing it) has not been employed for some time, von Bergmann having shown that healing frequently occurred under the first dry dressing, incision of the parts and drainage having been omitted.

Everyone should be acquainted with the general principles which control the application of the first dressing, for in this way severe infections (which so often develop in contused and lacerated wounds) may be prevented. The wound after the clothing is removed should be covered with dry aseptic gauze, or in case of emergency with fresh, clean linen. Some sort of a splint should then be applied to prevent displacement of the fragments. Hæmorrhage should be controlled by an Esmarch constrictor or by an appropriate bandage. It is exceedingly dangerous to manipulate a compound fracture, to sponge out or irrigate the wound, and to replace protruding fragments unless the aseptic arrangements are very complete.

Acute, progressive suppurative, and gangrenous infections of the soft tissues, pyogenic infections of exposed joints, necrosis of bone, and tetanus may be caused by the undue zeal of some good but ignorant Samaritan.

The patient, after being undressed, should be placed on an operating table. During and after the removal of the emergency dressing, the limb should be held by an assistant or assistants. The wound should then be carefully protected from the surrounding parts by sterile towels. If, after examination, a fracture is found, an anæsthetic should be administered. Then while the wound is protected by a sterile dressing, the surrounding area should be sterilized. The larger particles of foreign matter should then be removed with sterile forceps, the smaller by gentle irrigation of the wound with a three per cent solution of hydrogen peroxid or physiological salt solution.

After thorough cleansing of the wound the fracture is reduced, and if it is thought necessary the fragments should be held in place by a bone suture or some other device. Whenever it is indicated, counter-openings should be made to provide for drainage. The wound should then be loosely packed with iodoform gauze, which provides

capillary drainage. A plaster-of-Paris bandage should then be applied. A fenestrum should be cut in the cast over the wound through which the dressings can be changed when indicated. In favorable cases the wound may be partially sutured after the removal of the drainage.

It is a mistake to close wounds associated with compound fractures completely, for then an opportunity is afforded the bacteria which have been carried into the wound to develop. The mildest forms of infection may then become exceedingly virulent. Tetanus developed in two compound fractures which were closed by suture in von Bergmann's clinic.

Immediate amputation may be indicated if the soft tissues about the fractures are badly crushed, if the distal part of the extremity is anæmic and no pulse can be felt, rendering it probable that the principal artery has been destroyed. Occasionally, however, amputation is indicated later even when the limb can be saved, because of the contractures following the loss of large areas of skin and the laceration of the muscles. If an extremity is torn off from the trunk, the vessels should be ligated, the projecting fragments cut off, and crushed and contused tissues removed. In the treatment of compound fractures of the fingers and toes, it is advisable to attempt to save the projecting phalangeal fragment, and after the development of healthy granulation tissue, skin-grafting can be resorted to.

It may be necessary to incise phlegmons which develop during convalescence. If an osteomyelitis develops, good drainage should be provided, and the pieces of necrotic bone removed. Suppurative arthritis demands drainage of the joint, resection when the conditions indicate it. Amputation may be necessary because of a general infection or tetanus.

An attempt to improve the function of the part should be made after the infection has subsided. Extension apparatus, passive motion, and massage may be employed for this purpose. Malposition of the fragments and anchylosis are frequent when the patient is compelled to remain in bed for some time because of the poor condition of the wound. Not infrequently union is delayed in compound fractures and pseudarthrosis is common. This may be due to the extensive destruction of the periosteum, to infection, necrosis, or resection of the fragments.

LITERATURE.—*Albers, v. Bardeleben, Korsch.* Ueber Gehverbände. Chir. Kongr.-Verhandl., 1894, II, pp. 63–93.—*Bardenheuer.* Leitfaden der Behandlung von Frakturen und Luxationen der Extremitäten mittels Feder- resp. Gewichtsextension. Stuttgart, 1890.—*Bardenheuer* und *Grässner.* Die Behandlung der Knöchelbrüche mit Extensionsverbänden und die damit erzielten Resultate. Kölner Festschrift, 1904, p. 113.—

Bayer. Ueber Spiralbrüche an den oberen Extremitäten. Deutsche Zeitschr. f. Chir., Bd. 71, 1904, p. 204.—*Beely.* Zur Behandlung der einfachen Frakturen der Extremitäten mit Gips-Hanfschienen. Königsberg, 1878.—*v. Bergmann.* Erste chirurgische Hilfeleistungen an Verunglückten, in Meyers erste ärzliche Hilfe, Berlin, 1903.—*Bier.* Die Bedeutung des Blutergusses für die Heilung des Knochenbruches. Med. Klinik, Bd. 1, Heft 1, 1905.—*Bliesener.* Ueber die durch die Bardenheuersche Extensionsmethode an den Brüchen der unteren Gliedmassen exhaltenen funktionellen Ergebnisse. Deutsche Zeitschr. f. Chir., Bd. 55, 1900, p. 276.—*Blohm.* Ueber Vereiterung subkutaner Frakturen., I.-D., Berlin, 1898.—*P. Bruns.* Die Lehre von den Knochenbrüchen. Deutsehe Chir., Stuttgart, 1886.—*Bum.* Die Entwicklung des Knochenkallus unter dem Einflusse der Stauung. Arch. f. klin. Chir., Bd. 67, 1902, p. 652.—*Demisch.* Ueber Temperatursteigerungen bei der Heilung subkutaner Frakturen. I.-D., Zurich, 1885.—*v. Eiselsberg.* Zur Heilung grösserer Defekte der Tibia durch gestielte Hautperiostknochenlappen. Chir. Kongr.-Verhandl., 1897, II, p. 278;—Die heutige Behandlung der Knochenbrüche. Deutsche Klinik, Bd. 8, p. 521, Berlin, 1903.—*Flatu.* Muskelatrophien nach Frakturen, etc. Zentralbl. f. Grenzgeb., 1902, p. 305.—*Franke.* Behandlung komplizierter Frakturen. Arch. f. klin. Chir., Bd. 62, 1901, p. 680.—*Hähnle.* Die gerichtsärzliche Beurteilung schlecht geheilter Frakturen und Luxationen, wenn in Frage steht, ob Kunstfehler vorliegt. Deutsche Medizinalzeitung, 1903.—*Hänel.* Ueber Frakturen mit Bezug auf das Unfallversicherungsgesetz. Deutsche Zeitschr. f. Chir., Bd. 38, 1894, p. 129.—*Helferich.* Atlas und Grundriss der traumatischen Frakturen und Luxationen. München, Lehmann, 1903.—*Hoffa.* Lehrbuch der Frakturen und Luxationen. Stuttgart, Enke, 1904.—*Jordan.* Die Massagebehandlung frischer Knochenbrücke. Münch. med. Wochenschr., 1903, p. 1148.—*Jottkowitz.* Ueber Heilungsresultate von Unterschenkelbrüchen mit Bezug auf das Unfallversicherungsgesetz. Deutsche Zeitschr. f. Chir., Bd. 42, 1896, p. 610.—*Klauber.* Ueber komplizierte Frakturen der Extremitäten. Beitr. z. klin. Chir., Bd. 43, 1904, p. 319.—*Fritz König.* Ueber die Berechtigung frühzeitiger blutiger Eingriffe bei subkutanen Knochenbrüchen. Arch. f. klin. Chir., 1905.—*Kristinus.* Bericht über 130 Gehverbände. Wien. med. Wochenschr., 1902, No. 51.—*Loew.* Kondylenbrüche des Kniegelenkes. Deutsche Zeitschr. f. Chir., Bd. 44, 1897, p. 422.—*Lossen.* Grundriss der Frakturen und Luxationen. 1897.—*Lucas Championnière.* Traitement des fractures par le massage et la mobilisation. Paris, 1895;—Quelles sont les fractures qui peuvent être traitées sans appareil inamovible par le massage et la mobilisation? Résultats de ce traitement. Zentralbl. f. Chir., 1900, p. 1303. 13. internat. mediz. Kongress, Paris.—*Matas.* Remarks on some Controverted Questions in the Treatment of Fractures. Zentralbl. f. Chir., 1902, p. 777.—*W. Müller.* Ueber die heutigen Verfahren der Pseudarthrosenheilung. v. Volkmanns Samml. klin. Vortr., No. 145, 1896.—*Reichel.* Zur Behandlung schwerer Formen von Pseudarthrosis. Chir. Kongr.-Verhandl., 1903, II, p. 239.—*Riedinger.* Die ambulatorische Behandlung der Beinbrüche. Würzburg. Abhandl., 1902, Bd. 2, Section 9.—*Sudeck.* Zur Altersatrophie und Inaktivitätsatrophie der Knochen. Fortschr. auf dem Gebiete der Röntgenstrahlen, Bd. 3, 1900.—*Valenzuela.* Erfolge der Behandlung durch Bewegung und Massage in 61 Frakturfällen. Zentralbl. f. Chir., 1901, p. 666.—*v. Volkmann.* Die Behandlung der komplizierten Frakturen. Samml. klin. Vortr., 1877, Nos. 117–118.—Die Krankheiten der Bewegungsorgane. In Pitha-Billroths Handb. d. Chir.—*J. Wolff.* Ueber die Wechselbeziehungen zwischen der Form und der Funktion. Leipzig, 1901.—*Wolkowitsch.* Ueber die von mir angewandten Behandlungsmethoden der Brüche der grossen Extremitätenknochen, etc. Deutsche Zeitschr. f. Chir., Bd. 63, 1902, p. 203.—*Ziegler.* Ueber das mikroskophische Verhalten subkutaner Brüche langer Röhrenknochen. Deutsche Zeitschr. f. Chir., Bd. 60, 1901, p. 201.

X. INJURIES OF BODY CAVITIES AND DIFFERENT VISCERA

These injuries may be subcutaneous or open; naturally the two forms are frequently combined.

Subcutaneous injuries are produced by blunt force, such as a fall or a blow. In many cases the symptoms are those of internal hæmorrhage. The symptoms may differ a great deal, depending upon the organs or organ injured.

Hæmatomas of the cranial cavity following lacerations of the meningeal arteries or the cranial sinuses give rise to most serious symptoms. Hæmorrhage from the middle meningeal artery, associated with a fracture of the skull or contusions, is especially dangerous, as the blood forces its way between the dura mater and the skull, giving rise to the symptoms of brain pressure which is often followed by death.

Subcutaneous injuries of the abdomen may be associated with the laceration of large vessels or the rupture of a vascular viscus. So much blood is then lost rapidly, unless the hæmorrhage is checked, that death soon occurs. The dangers of hæmorrhage into the chest cavity are not so great as those of pressure exerted by the extravasated blood, except when the heart is ruptured. In injuries of the chest hæmorrhage may occur from the intercostal and internal mammary arteries, from the lung which has been pierced by a fractured rib, or from some of the larger vessels about the heart.

After very forceful compression of the abdomen and thorax, an extravasation of blood into the tissues of the neck and head, associated with disturbances of vision, may occur. According to Perthes, Braun, and Sick the extravasation of blood in these cases is due to a sudden increase of pressure in the capillaries with subsequent rupture of the same as the result of the rapid increase in intrathoracic pressure. In these cases it is probable that the lungs are distended and that the glottis is involuntarily or reflexly closed, thus preventing escape of air from the lungs when the pressure is exerted.

Of the subcutaneous injuries, those of the brain, such as are associated with injuries and fractures of the skull, are the most important. Contusions and lacerations of the spinal cord accompany fractures of the vertebræ. Laceration of the lung and rupture of the heart may be associated with fractures of the ribs. The same lesions may occur, however, without fracture of the bones surrounding these organs. Subcutaneous injuries of the abdomen may be associated with rupture or extensive laceration of the liver and spleen followed by fatal hæmorrhage, or with rupture of the stomach and intestine followed by a rapidly developing fætal peritonitis. The urinary bladder is readily ruptured when full, if a blow is delivered upon the abdomen. In

fractures of the pelvis the bladder may be lacerated by the displaced fragments of bone. The kidney may be torn, lacerated, or completely separated from its pedicle as a result of a subcutaneous injury of the abdomen.

Open wounds of the body cavities are most frequently caused by knives, stilettoes, or projectiles; occasionally by sharp pieces of iron, pickets, canes or umbrellas, axes and scythes. Hæmorrhage and infection, which latter is carried in with the vulnerating force or develops after rupture of a hollow viscus, are the principal dangers. Extensive open injuries are often caused by explosions.

The symptoms, diagnosis, and treatment of these various injuries are fully discussed in text-books devoted to special surgery.

LITERATURE.—*v. Bergmann, v. Bruns, v. Mikulicz.* Handb. d. prakt. Chir. Enke, Stuttgart, 2. Aufl.—*König.* Lehrbuch der speziellen Chirurgie. Hirschwald, Berlin, 8. Aufl., 1904.—*Milner.* Die sog. Stauungsblutungen infolge Ueberdruckes im Rumpf. Deutsche Zeitschr. f. Chir., Bd. 76, 1905, p. 85.—*Perthes.* Ueber ausgedehnte Blutextravasate am Kopf infolge von Kompression des Thorax. Deutsche Zeitschr. f. Chir. Bd. 50, 1899, p. 436;—Ueber Druckstauung. Ebenda, Bd. 55, 1900, p. 384.

XI. GUNSHOT WOUNDS

Gunshot wounds demand special consideration. They may be caused by firearms of small caliber or by artillery.

WOUNDS CAUSED BY FIREARMS OF SMALL CALIBER

Small shot and ordinary bullets are made of either soft or chilled lead and are round, conical, or shaped like an acorn. The bullets used in the army are made of chilled lead, and are completely or partially jacketed with steel or some other metal. Rifles used at the present time are of much smaller caliber (0.25″–0.31″) than those formerly employed (0.44″–0.50″).

Wound of Entrance and of Exit—Wound Canal.—Bullet wounds have a wound of entrance, a canal, and if the projectile perforates, a wound of exit. The wound of exit is usually larger than the wound of entrance, and has irregular, jagged outlines. The wound canal may be straight. The projectile when partially spent may be deflected by bone or resistant tissue, and then the canal will be curved. The diameter of the canal corresponds to the diameter of the projectile. The canal near the wound of exit may be funnel-shaped. Under certain conditions the tissues surrounding the canal are lacerated. This is especially apt to be the case when the projectile has a high explosive force. There may be multiple wounds when a bone is comminuted and the fragments are driven through the skin, or when the projectile explodes.

The form of a gunshot wound depends upon the character of the projectile, the velocity with which it travels, and the range at which the projectile is fired. Soft projectiles which are altered in form or split into a number of fragments by bone or resistant tissue cause more extensive wounds than hard projectiles or those which are partially or completely jacketed.

Jacketed bullets do the most damage when fired at a range of about 200 yards, for at this distance the small caliber projectile has a high initial velocity and great potential force. Bullets made of soft lead or soft compounds which permit of mushrooming when they come in contact with solid or resistant tissue lacerate the tissues, producing enormous cavities and large wounds of exit. Because of this mutilating action the use of the soft-nosed or Dum-Dum bullet and of the hollow-

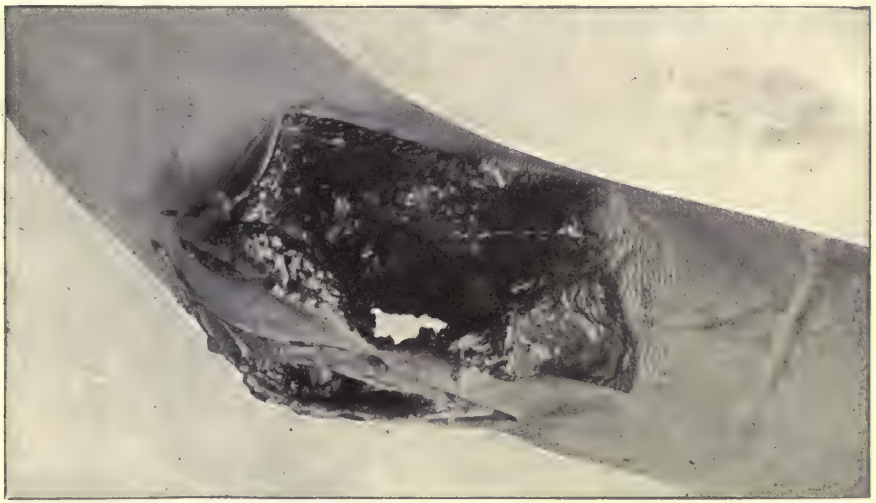


FIG. 229.—DESTRUCTION OF THE ELBOW JOINT CAUSED BY A LEAD-POINTED (DUM-DUM) BULLET FIRED AT CLOSE RANGE.

nosed bullet has been prohibited by the International Peace Congress meeting at The Hague. The form of even a completely jacketed bullet may be altered by striking a rock or some other very hard object before it enters the body.

Powers of Penetration.—The penetration depends upon the initial velocity of the projectile and upon its potential force and hardness. Small pointed projectiles have greater penetrating force than large round ones. The full-jacketed projectile used in modern rifles has a penetrating power which enables it to pass through a number of bodies when fired at a range of from 600 to 1,000 yards. It therefore penetrates more deeply, lodges less frequently, causes much less damage, and

has smaller wounds of entrance and exit and a narrower canal than the lead bullets which were formerly used.

Frightful wounds are associated with gunshot injuries of bones and of those organs which contain fluids or of encapsulated tissues rich in fluids, such as the skull, the heart, the full stomach and intestines, the liver, spleen, and kidney. The greatest damage is probably inflicted when the projectile is fired at close range and has a high initial velocity. The minimum of damage is inflicted by a projectile with a medium velocity, while a bullet which is well spent inflicts a little more.

The symptoms of a gunshot wound naturally depend entirely upon the tissues or the viscera which are injured. Gunshot wounds of the heart, brain, and large vessels are usually fatal, death occurring immediately or soon after the reception of the wound.

Symptoms of a Gunshot Wound.—The first symptom is usually a short sharp pain. The pain associated with wounds inflicted by projectiles of small caliber may be very slight, and even in gunshot wounds of the

FIG. 230.—ROENTGEN-RAY PICTURE OF FIG. 229.

abdomen and chest it is frequently compared to the pain associated with a wound produced by a small stone. The impact is more painful when a bone, joint, or nerve is injured. A gunshot wound of the brain causes unconsciousness, of the spinal cord paralysis. Sensation is rapidly lost in the tissues about a gunshot wound. In severe injuries the part of the body involved may be cool, pale, incapable of motion, and

without sensation. This local wound shock may pass over into general shock.

Hæmorrhage.—The amount of hæmorrhage varies. The smaller the cutaneous wound and the narrower the canal, the less marked the external hæmorrhage will be. The canal is quickly closed by clots, and besides the tension planes in the tissue differ, so that when the tissues are divided the canal is quickly closed by their overlapping. The amount of external hæmorrhage may be small, even when large vessels are injured. In such a case, however, an extensive hæmatoma may form in the soft tissues and in the body cavities, and even after the injury of a small vessel the hæmorrhage may be great enough to cause death. The elastic vessels were often pushed aside and escaped injury when the old-style lead bullets were used. The modern small steel-jacketed bullet passes directly through the artery or cuts it off, and is very apt to cause severe hæmorrhage (von Bergmann). When the wound is small internal hæmorrhage is much more to be feared than external hæmorrhage (Küttner).

Varieties.—*Gunshot Wounds of the Skin.*—Gunshot wounds of the skin present the greatest variety. A spent bullet causes merely a contusion of the skin. If the bullet strikes the surface of the body vertically the wound of entrance will be round. It will correspond in size to the caliber of the bullet. A wound caused by a steel-jacketed bullet is smaller than the projectile, as the pointed nose of the bullet makes the skin tense before it perforates. The margins of the wound are more or less contused, and frequently the wound itself is funnel-shaped. If the skin lies directly over bone the margins of the wound may be everted, and in this case the wound will be larger than when soft tissues intervene between the skin and bone. If the projectile has been fired at very close range, the skin surrounding the wound will be burned and filled with deeply imbedded grains of powder, the removal of which is very painful. Some of the superficial grains may be discharged if an artificial inflammation of the skin is produced by a one per cent sublimate solution (Hebra). If the weapon is fired at very close range, as in attempts at suicide, the skin or mucous membrane may be badly lacerated by the gases formed when the powder explodes. In a simple gunshot wound of the soft tissues the wound of exit is similar to the wound of entrance. When, however, the explosive action is great, the wound of exit is much larger than the wound of entrance, and has notched, irregular, and undermined edges beneath which lie shreds of tissue and splinters of bone. Large gaping wounds of exit are caused by lead bullets of large caliber and by partially jacketed bullets fired at ranges varying from a few feet up to 200 yards.

If the projectile strikes at an angle, the wounds of entrance and exit

will be large and oval or irregular, if the form of the projectile has already been altered by striking some hard object. Multiple wounds of entrance and exit may be produced by the modern small-caliber projectile if it perforates different parts of the body, cutaneous folds, or muscular prominences.

A projectile striking the body tangentially produces long, grooved wounds, the edges of which are undermined. If the bullet passes just beneath the skin, the course of the wound canal will be indicated by ecchymoses.

If the bullet is perfect the canal will be smooth and narrow, corresponding in size to the diameter of the former. If the bullet is fired at short range, if its form is altered by coming in contact with bone, or if its explosive force is great, the tissues along the wound will be extensively lacerated and the wound of exit will be large.

Injuries of Vessels.—Contusion of vessels with subsequent necrosis of their walls occurs in both penetrating and perforating gunshot wounds. Small arterial wounds caused by jacketed bullets are much like other wounds of the arterial wall and end in scar formation with complete healing or in the development of an aneurysm. An arteriovenous aneurysm may develop if the projectile passes between an artery and a vein, injuring corresponding parts of the walls of both vessels.

Injuries of Nerves.—Complete division of peripheral nerves is more frequently caused by large lead bullets than by the small jacketed ones. The latter may penetrate a nerve, merely making a slit in it, even when the nerve is of the same diameter as the bullet. The symptoms of an incomplete paralysis which follow such an injury disappear after a few weeks. Neuralgia and neuritis are frequently caused by a bullet lodged immediately adjacent to a nerve.

Injuries of Bones.—A bone may be contused, fractured, or perforated by a projectile. A soft-lead bullet, the form of which is easily altered, when traveling with but little velocity becomes flattened out when it strikes bone and causes merely a contusion, associated with an extravasation of blood beneath the periosteum and into the bone marrow. Occasionally the projectile does not perforate the skin, merely contusing the latter and producing a subcutaneous fracture.

Jacketed bullets fired at close range perforate spongy bones and the ends of long bones. If the projectile passes at right angles to the bone, it produces a straight canal, the wound of exit being larger than the wound of entrance. If it strikes tangentially, a groove is formed in the bone. The large soft-lead bullets formerly used comminuted and fissured the bones and lodged in the spongiosa, lacerating it. They rarely perforated the bone.

The diaphyses of long bones are brittle and may be comminuted by both the jacketed and soft-lead projectiles. If the projectile is fired at close range the fragments are smaller than when it is fired at long range, but the extent of the area comminuted is the same (Fig. 229). The typical fracture when the comminution is great is the " butterfly " fracture, in which two lateral fragments (the wings of the butterfly)

FIG. 231.—FRACTURES OF THE DIAPHYSIS OF THE HUMERUS CAUSED BY A JACKETED BULLET. (After Küttner.) *a*, Close range (up to 180 yards); *b*, long range (1,000–2,000 yards.)

are separated by two fissures passing upward and downward on each side of the wounds of entrance and exit. If the projectile does not pass through the greatest diameter of the bone, the resemblance of the fracture to the form of a butterfly is not very striking, as the four fissures have an irregular course, the fragments are smaller, and are

displaced more (Fig. 230). Fissures associated with these fractures frequently extend into the adjacent joints. Oblique and transverse fractures may be caused by projectiles grazing a bone.

Relation of Destructive Action to Range.—The closer the range the greater will be the laceration of the tissues surrounding the tract of the projectile. Large cavities filled with blood, fragments of soft tissue, and splinters of bone will then be found. The cavities behind the perforated bone will be larger than those in front of it. If no soft tissues intervene between the bone and the skin, the wound of exit is

a *b*

FIG. 232.—GUNSHOT WOUND OF THE LOWER END OF THE RIGHT HUMERUS (1866). *a*, Wound of entrance on posterior surface; *b*, wound of exit anterior. (Amputation performed by von Langenbeck.)

frequently quite large, corresponding in size to the cavity beneath the skin which is filled with detritus. If soft tissues intervene between the bone and the skin, the wound of exit will be of about the same size as the wound of entrance. The laceration and the formation of cavities are not so marked when the projectile (steel-jacketed bullet fired at a range of from 1,600 to 2,000 yards) is fired at long range, for in these cases the fragments are not so widely separated, and some of them remain attached to the periosteum.

Gunshot Wounds of Joints.—Gunshot wounds of joints are almost always complicated by fractures of the articular ends of the bones enter-

ing into the formation of the joint involved. Lead bullets almost always cause extensive comminution of the bones (Fig. 230). The small lead bullets discharged from the ordinary pistol do not have much penetration, and frequently they become imbedded in the ligaments or enter the joint cavity without injuring the bones.

Gunshot Wounds of Body Cavities and Viscera.—Of gunshot wounds of the body cavities and viscera, those of the skull and its contents are

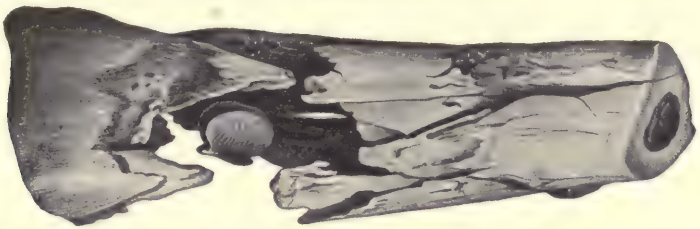


FIG. 233.—COMMINUTED FRACTURE OF THE TIBIA (Battle of Schleswig, 1848). Resection performed by B. von Langenbeck.

the most dangerous. Shots fired at close range are almost always fatal because of the explosive force of the projectile, which is especially marked in small-caliber projectiles used in warfare at the present time. The brain is reduced to a bloody pulp, and occasionally when the shot is fired at very close ranges (e. g., in attempts at suicide) the pulpy

FIG. 234.—GUNSHOT WOUND OF HEAD OF THE HUMERUS (Battle of Düppel, 1848). Splintered fracture of the head of the humerus. Long head of the biceps divided by a fragment of bone. Bullet imbedded in the bone. Preparation made after resection by B. von Langenbeck.

mass is driven out of the wound of exit (Krönlein's "exenteratio cranii") and the skull is broken up into a countless number of splinters. Projectiles fired at middle or long range, or those traveling with but little velocity, may fracture or penetrate the skull and become lodged in the brain. If there is no hæmorrhage or infection, and the part of the brain injured does not control some vital function, such a bullet

may become encapsulated and cause but little or no interference with the functional activity of the organ.

Gunshot wounds of the abdomen are especially dangerous, as the projectile may lacerate a vascular organ, causing a fatal hæmorrhage, or may open the stomach, intestine, or some other hollow viscus, causing a fatal peritonitis. Spontaneous healing of wounds of the intestinal tract occasionally occurs as the result of the adhesions forming about the opening or of agglutination between the tissues surrounding the opening and those immediately adjacent to it. Spontaneous healing occurs more frequently after wounds caused by small steel-jacketed bullets than after those caused by large lead bullets. [In penetrating gunshot wounds of the abdomen an immediate laparotomy should be performed. It is dangerous to delay operation, and the expectant treatment should be discouraged.]

Prognosis.—The prognosis of gunshot wounds of the chest is good, except when the heart or great vessels are injured. A number of patients, however, die later as the result of suppurative or putrefactive inflammation of a hæmothorax. Wounds of the lungs caused by projectiles heal rapidly. Internal hæmorrhage is to be feared only when the larger pulmonary vessels are injured, or when the lung is torn by a fragment of the rib carried in by the bullet or by a deformed projectile.

WOUNDS CAUSED BY ARTILLERY

Wounds produced by artillery are much worse than even the most terrible wounds caused by projectiles of small caliber when fired at close range.

Cannon balls, exploding bombs and shells, and shrapnel are discharged by artillery. The exploding shells are filled with hollow projectiles, and when they explode a very large number of small projectiles are hurled in every direction. The velocity with which these small projectiles travel varies from 450 to 1,000 yards per second. Shrapnel contains from 300 to 500 hard-lead bullets weighing 10 gm. each. These are discharged when the shell strikes or explodes during its flight and have a greater power of penetration than the fragments of the shell itself. The explosion is governed by a time fuse, and the shell can be made to explode in front of or above its mark.

Bombs and the so-called indirect projectiles (fragments of stone or splinters of wood hurled when the projectile strikes or explodes) cause extensive lacerations and contusions which may be seen about the wounds of entrance, but are especially marked about the wounds of exit. Pieces of clothing are frequently found in the irregular, ragged wounds of entrance, and shreds of muscle, lacerated tendons, and splinters of bone

project from the wounds of exit. The amount of injury varies with the size of the projectile and the velocity with which it travels. The body cavities may be opened widely or an extremity torn from the trunk by a large projectile traveling with great velocity; a contusion or a groove may be produced by a spent bullet.

Fatal internal injuries, deafness, and concussion of the brain may be caused by the explosion of a shell near by an individual, even when he is not struck by any of the fragments. The explosive force may be great enough to hurl him against a tree or wall causing open and subcutaneous injuries.

Wounds caused by projectiles fired from artillery are more frequently fatal than are those caused by projectiles of small caliber, and are more frequently accompanied by shock and severe hæmorrhage. The dangers are also increased by the size of the wounds as the dangers of infection are much greater.

CLINICAL COURSE OF GUNSHOT WOUNDS IN GENERAL

The clinical course of a gunshot wound depends, leaving out of consideration hæmorrhage, upon the position of the wound and its severity, upon whether infection develops or not.

Primary infection is to be feared but little, although there is always abundant opportunity for the introduction of bacteria.

It has been determined by animal experiments that a bullet may carry infection from the clothing and skin into the deeper tissues. An animal may develop a fatal infection from highly virulent bacteria attached to a bullet (A. Müller, Koller). Clinical experience, however, has shown that the infection (unless introduced secondarily) associated with bullet wounds is rarely virulent, and that the bacteria carried in by bullets are readily destroyed by the bactericidal substances in the tissue fluids. Even particles of clothing may be encapsulated in the sensitive lining of a joint without causing suppuration. Clinically the majority of bullet wounds are to be regarded as not infected, unless secondary infection is introduced by probing and improper and ill-advised treatment. Wounds caused by blank cartridges are an exception to this rule, as the wads which are shot into the tissues are made of rags and contain in many instances virulent tetanus bacilli.

Secondary Infection.—Secondary infection of bullet wounds is the greatest danger. It occurs most frequently in wide, gaping wounds. The smaller the wounds of entrance and exit, the less the dangers of secondary infection and the more rapid the repair. Probing for the bullet, exploration of the wound canal, and irrigation of the wound with antiseptic solutions are dangerous, for bacteria may be carried from the margins of the wound into the wound canal, and the resistance

of the tissues to bacterial invasion may be reduced. Almost all gunshot wounds of bones and joints treated by the earlier methods of probing, irrigation, and exploring the wound canal were associated with suppuration, general infections claiming many victims if early amputations were not performed. Von Bergmann was the first to advise a rational method for the treatment of gunshot wounds. He demonstrated during the Russo-Turkish war that the most dangerous injury—a wound of the knee joint—would heal without infection if after sterilization of the skin surrounding the wound a dry aseptic dressing were applied.

Diagnosis and Treatment.—The diagnosis is based upon the symptoms associated with injuries of the different viscera or tissues. An immediate laparotomy should be performed in gunshot wounds of the abdomen, if it is probable that the projectile has penetrated the abdominal wall. Bullet wounds of the chest should not be operated upon unless there is some indication. A Roentgen-ray picture may be required to determine the extent and type of an injury to a bone or the position of the bullet.

In the treatment an attempt should be made to prevent secondary infection, and to provide conditions favorable for repair. The first-aid dressing is very important in preventing secondary infections. The wound should be covered as quickly as possible with sterile gauze. Each soldier at the present time is provided with an emergency dressing which he can apply himself. This dressing is so made that it can be easily applied without the fingers coming in contact with the layer of gauze resting upon the wound. In hæmorrhage from large vessels of the neck and trunk which cannot be controlled by an Esmarch constrictor, it is of vital importance to control the bleeding, and in such cases, and in these only, can the aseptic dressing of the wound be neglected.

The definitive dressing is carried out according to the principles already given. The wound is protected by sterile gauze while the surrounding skin is being shaved and sterilized. Large foreign bodies are then removed with the tissue forceps, small particles by gentle irrigation with physiological salt solution or a three per cent solution of hydrogen peroxid. Shreds of tissue and loose splinters are removed from large wounds, spurting vessels are caught and ligated, and counter-openings are provided for drainage if indicated. Crusts forming on small wounds should be allowed to remain, unless there is some indication for removal.

Every gunshot wound should be treated by the open method—that is, it should not be closed by plaster or sutures. Dry aseptic gauze serves a double purpose as a dressing for these wounds: (1) It absorbs secretion; (2) it protects the wound from outside infection. By this dressing the small wounds of entrance and exit, and even the larger wounds

situated in cavities filled with lacerated tissue, fractured bones, and injured joints, are, as it were, transformed into simple, subcutaneous wounds. This is not the case in very large wounds. In some of these it is not always possible to prevent severe infections even when all the recesses are carefully tamponed and good drainage is provided. In these cases phlegmons may develop; secondary hæmorrhages may occur from injured blood vessels; infections of bones and joints, or tetanus may develop. If these complications develop, incisions, resections, amputations, and ligations should be performed when indicated. Amputations are more frequently performed in military than in civil practice. Time and facilities, which render successful conservative treatment possible, are often wanting during the stress of battle. The experiences of the Russo-Turkish, the Boer, and Spanish-American wars indicate that there is a decreasing demand for mutilating operations in the treatment of gunshot wounds.

Bullets, fragments of stones, splinters of wood, etc., if they are seen in the wound or are felt directly beneath the skin, should be removed. They should also be removed if an infection preventing encapsulation develops, if they cause pain or interfere with function after they have become encapsulated.

If possible the wounded part should be put at rest by an immobilizing dressing, so that wound repair will not be interfered with by movements. Naturally, immobilization should be employed when bones are fractured, but it is also of value in treating gunshot wounds of tendon sheaths and of muscles.

Laparotomy, combined with intestinal suture if the gastrointestinal tract is perforated, and trephining of the skull to remove splinters of wood, elevate a depressed fragment or to control hæmorrhage are indicated in gunshot wounds of the abdomen and skull. A kidney or the spleen may be so badly lacerated that extirpation of the injured viscus is indicated. As a rule, the expectant treatment is more frequently employed in military than in civil practice, as the conditions existing in the former do not always permit of extensive operative interference, and besides, spontaneous healing occurs more frequently in wounds caused by projectiles of small caliber that are used in war.

The first-aid treatment consists of covering the wound with sterile gauze; in applying an Esmarch bandage to control hæmorrhage; of supplying stimulants when the patient is suffering from shock or has lost consciousness, and of introducing a tracheotomy tube or a tracheal canula after injury of the larynx or trachea (von Bergmann).

The first dressing should be applied upon the battlefield, where, in addition to adjusting permanent dressings and preparing the wounded for transportation, more serious operations, such as the ligation of arter-

ies and amputations, may be performed when indicated. It may also be necessary to perform a urethrotomy in gunshot wounds of the perineum and pelvis or a tracheotomy in gunshot wounds of the neck when there is interference with breathing (von Bergmann). All other operations should be performed in the field hospitals, of which each army corps has twelve with accommodations for 200 each (Schjerning).

LITERATURE.—*v. Bergmann.* Die Behandlung der Schusswunden des Kniegelenks im Kriege. Stuttgart, 1878;—Erste Hilfe auf dem Schlachtfelde und Asepsis und Antisepsis im Kriege. Aerztl. Kriegswissenschaft, Jena, 1902.—*v. Bruns.* Ueber die kriegschirurgische Bedeutung der neuen Feuerwaffen. Chir. Kongr.-Verhandl., 1892, I, p. 1;—Inhumane Kriegsgeschosse. Chir. Kongr.-Verhandl., 1898, II, p. 317;—Ueber die Wirkung der Bleispitzengeschosse. Beitr. z. klin. Chir., Bd. 21, 1898, p. 825. —*v. Coler* und *Schjerning.* Ueber die Wirkung und kriegschirurgische Bedeutung der neuen Handfeuerwaffen. Mediz. Abteil. des kgl. preuss. Kriegsministeriums, 1894.—*Fischer.* Handbuch der Kriegschirurgie. Deutsche Chir., 1882.—*Flockemann, Ringel, Wieting.* Kriegserfahrungen aus dem südafrikanischen Kriege. v. Volkmanns Samml. klin. Vortr., 1901, Nos. 295–296.—*Hildebrandt.* Zur Erklärung der Explosionsschüsse. Münch. med. Wochenschr., 1903, No. 25, p. 1061;—Zur Erklärung der Bewegungsvorgänge bei Explosionsschüssen. Arch. f. klin. Chir., Bd. 72, 1904, p. 1050.—*Kayser.* Experimentelle Studien über Schussinfektion. Beitr. z. klin. Chir., Bd. 26, 1900, p. 282. —*Kocher.* Zur Lehre von den Schusswunden durch Klienkalibergeschosse. Kassel, 1895. —*R. Köhler.* Die modernen Kriegswaffen. Berlin, 1897.—*König.* Schussverletzungen am Rumpfe, insbesondere am Thorax. Aerztl. Kriegswissenschaft, Jena, 1902.—*Kranzfelder* und *Schwinning.* Die Funkenphotographie, insbesondere die Mehrfachfunkenphotographie in ihrer Verwendbarkeit zur Darstellung der Geschossiwirkung im menschl. Körper. Mediz. Abteil. d. kgl. preuss. Kriegsministeriums, Berlin, Juni, 1903.—*Küttner.* Kriegschirurgische Erfahrungen aus dem südafrikanischen Kriege, 1899–1900. Beitr. z. klin. Chir., Bd. 28, 1900, p. 717.—*Mohr.* Schussverletzungen durch kleinkalibrige Gewehre, speziell nach den Erfahrungen der letzten Feldzüge. Arch. f. klin. Chir., Bd. 63, 1901.—*Pirogoff.* Grundzüge der allgemeinen Kriegschirurgie. Leipzig, 1864.—*Reger.* Ueber die kriegschirurgische Bedeutung der neuen Feuerwaffen. Chir. Kongr.-Verhandl., 1892, II, p. 19;—Die Krönleinschen Schädelschüsse. Ibid., 1901, II, p. 508.—*Schjerning.* Die Organisation des Sanitätsdienstes im Kriege. Aerztl. Kriegswissenschaft, Jena, 1902, p. 229;—Ueber die Bekämpfung des Tetanus in der Armee. Veröffentl. aus d. Geb. d. Militärsanitätsw. Berlin, 1903, Heft 23.—*Schjerning, Thöle* und *Voss.* Die Schussverletzungen. Fortschr. auf d. Geb. d. Röntgenstrahlen. Ergänzungsband 7, 1902.—*Schloffer.* Ueber embolische Verschleppung von Projektilen. Beitr. z. klin. Chir., Bd. 37, 1903, p. 698.—*Seydel.* Lehrbuch der Kriegschirurgie. Stuttgart, 1905.—*Skrzeczka.* Aus der gerichtsärzlichen Praxis (Platzwunden). Vierteljahrsschr. f. gerichtsärztl. Mediz., Bd. 10.

II. CHEMICAL INJURIES

Chemical substances which by their action cause death and degeneration of tissues are called caustics. They are frequently employed in surgery. Alkalies, such as sodium, potassium, and calcium; acids, such as hydrochloric, sulphuric, nitric, arsenious, chromic, and carbolic; and the salts of some metals, such as silver nitrate, zinc chlorid, and copper sulphate, are the most common types of caustics. The alkalies and metallic salts act by uniting with the albumens in the tissues; the acids by burning the structures with which they come in contact.

Action of Caustics.—Dilute and mild caustics, when applied, cause an inflammatory reaction. An erythema and vesicles develop, and the clinical picture of the lesion corresponds to that of a burn of the first or second degree. Necrosis followed by eschar formation is caused by strong caustics, the necrosis involving tissues at various depths, depending upon the strength of the caustic and the time it is allowed to act. The skin is less easily destroyed by caustics than is mucous membrane.

Acetic acid always has a superficial action, while the alkali caustics penetrate more deeply. Various caustics, such as arsenious, chromic, and lactic acids, ammonia, and copper sulphate, act only upon mucous membranes. Zinc chlorid has no effect upon healthy skin.

Those agents are best suited for cauterization which, like concentrated sulphuric and fuming nitric acid and silver nitrate, penetrate to the deeper layers of the skin and mucous membranes only after acting for some time. For this reason nitric acid is frequently applied to superficial hæmangiomas, while exuberant and diseased granulation tissue is destroyed by silver nitrate. Chromic acid is recommended by Czerny for the treatment of inoperable, malignant tumors, the ulcerated surfaces being covered with gauze saturated with from a twenty to fifty per cent solution. Lactic acid (fifty to eighty per cent) is frequently employed in the treatment of tuberculosis of mucous membranes.

Symptoms of Cauterization.—The pain following the application of a caustic is sometimes transitory (e. g., after nitric acid or silver nitrate); at other times it persists for some time (e. g., after concentrated carbolic acid and caustic calcium). After a caustic is applied a white, yellowish brown, or brown spot develops which, upon the skin, rapidly

becomes transformed into a dry hard crust; upon the mucous membranes into a discolored soft one. Gradually this crust or eschar is separated from the healthy surrounding parts by granulation tissue. Naturally a superficial eschar is separated more rapidly than a deep one. Several weeks may be required for the separation of a layer of bone which has been killed by a caustic. Severe hæmorrhage may occur during the separation of an eschar, for the walls of a large vessel may have undergone necrosis.

In Fig. 235 is represented the face of a man over whom caustic soda was thrown. He was brought to the clinic with large yellow crusts covering the entire face, both ears, the forehead, and temples. These crusts separated in two weeks, good healthy granulation tissue which was skin-grafted having developed. After a number of weeks several sequestra were discharged from the frontal bone. Granulation tissue which covered the remains of the bulb of the eye developed rapidly from the remnants of the left eyelid. This tissue was soon covered by epithelium which developed from that still attached to the eyelids. An open space in which an everted conjunctiva may be seen indicates the position of the right palpebral fissure.

Fig. 235.

Scars Following Cauterization.—The scars which remain after superficial cauterization are smooth and soft. Scars following the separation of deep eschars of the skin are inclined to the formation of keloidlike growths, and to cause the distortion of the parts which in the face may lead to narrowing of the natural orifices or to ectropion of the eyelids and lips.

Cauterization of the œsophagus and urethra may be followed by serious results, as the masses of scar tissue which develop may narrow or occlude their lumina. Acids and lye swallowed accidentally or taken with suicidal intent burn the stomach as well as the œsophagus. If perforation ending fatally does not occur, the scar tissue which develops later may narrow the œsophagus or cause a stenosis of the car-

diac and pyloric openings of the stomach. Injuries of the urethra are caused most frequently by the use of strong solutions of acids or silver nitrate in attempts to abort a beginning gonorrhea.

The nature of an eschar of the skin following cauterization is usually readily recognized. The reaction of the reagent can be determined in recent cases by the use of litmus paper. The statement of the patient is of value in determining the nature of the caustic, except in hysterical patients who not infrequently deliberately injure themselves.

Treatment.—The treatment of chemical injuries in recent cases should be directed toward neutralization of the agent before it penetrates deeply. Chemists and apothecaries are most frequently injured, and they usually have two solutions ready. Acetic acid or vinegar is used to neutralize the alkalies, while a solution of sodium bicarbonate is used to neutralize the acids. In some cases these agents are employed too late, the damage having already been done by the caustic.

If the tissues have already been destroyed, nothing can be done but to hasten the separation of the eschar by moist dressings, or to lessen the pain and prevent infection by the use of ointments. Cicatricial stenosis of the œsophagus, pylorus, and urethra should receive appropriate surgical treatment. Large granulating wounds should be skin-grafted.

LITERATURE.—*Arnd.* Aetzmittel, Aetzwunden. Kochers Realenzyklopädie. *Sonnenberg.* Verbrennungen and Erfrierungen. Deutsche Chir., 1879, ätzende Stoffe, p. 13.

III. THERMAL INJURIES

CHAPTER I

FREEZING

Cold acting upon the body produces local and general changes which are grouped under the term of freezing. The degree of the pathological changes depends upon the character and degree of the cold, the length of time that it acts, and upon the resistance of the organism or the part involved.

It is a well-known fact that moist cold acts more rapidly and severely than dry cold, and that a much lower degree of cold can be borne when there is no wind than when it is blowing strongly. Powerful healthy men are more resistant to cold than are weak, anæmic individuals and children and old people.

General Freezing.—*Symptoms.*—General freezing begins with severe and repeated rigors and a feeling of lassitude, gradually passing into a desire to sleep which cannot be resisted. If the individual cannot resist this desire to sleep, he falls in the snow or ice and dies. Drunken persons, children and old people, and anæmic individuals succumb most frequently and rapidly to freezing. If the individual has strength enough to resist the desire to sleep, he may keep on his way until he finds shelter or is rescued by friends or passers-by. As freezing progresses the individual reels like a drunken man, the senses become numbed, consciousness is lost, the pulse and respiration are slowed. Finally he collapses and rapidly dies of cardiac weakness and cerebral anæmia, or remains alive for a longer time (sometimes for days), eventually dying, the entire body becoming stiff, as the result of the freezing of the tissue fluids.

If the rectal temperature of the frozen individual is not below 68° F. when he is found, there is a possibility of resuscitating him, even if his cardiac and respiratory functions are weak. The convalescence, however, is usually slow in these cases, and is often accompanied by nervous symptoms (headache, loss of consciousness, delirium, paralysis). Death may occur even when convalescence seems to be fairly well established,

as the result of degenerative changes in the blood cells and cardiac weakness.

Treatment.—In the treatment, the results of which are always doubtful, an attempt should be made to restore gradually warmth to the frozen body and to aid the circulation. A sudden change into a warm room is always dangerous, because of the damage done to the tissues by thawing them out rapidly and because of the sudden entrance of a large number of degenerating red blood corpuscles into the circulation.

The frozen individual should not be brought immediately into a heated room, but should be detained in a cold room, the temperature of which registers 34° or 35° F. The entire body should be rubbed with snow or cold cloths in order to stimulate the cutaneous blood vessels. Camphorated oil should be injected to stimulate the heart, and the respiratory activity should be aided by artificial respiration carefully induced. The patient should then be placed in a bath of a temperature of 60° F. This temperature should be gradually raised within three hours to 86° F. As soon as the patient can swallow he should be given warm, stimulating drinks and nourishment. Frequently morphin is required to control the pain in the frozen part.

Local Freezing.—Local freezing affects most frequently those parts which are not protected by clothing and those in which the circulation is interfered with by tight and constricting articles of wearing apparel (gloves and boots). The ears, nose, cheeks, fingers, and toes are most frequently frozen.

Degrees of Frostbites.—Local freezing (frostbite) is usually divided into three degrees, depending on the severity, but often a sharp distinction between the different degrees cannot be made.

The first degree is characterized by a transitory hyperæmia, the second by the formation of vesicles, and the third by gangrene. Fremmert and Luppian divide the third degree into three subdivisions, depending upon the extent of the gangrene (gangrene of the superficial layers of the skin, of the cutis and subcutaneous tissues, and of all the tissues down to the bone). If we accept this classification, we must recognize five degrees of local freezing or frostbite. Chilblains form a peculiar chronic form of local freezing.

(*a*) *First Degree of Frostbite.*—The first-degree frostbite is the most common and mildest. It follows relatively short exposure to severe cold and is characterized by erythema and swelling, which develop as soon as the frozen part is warmed. The frozen part loses sensation, after a slight preliminary pain, and becomes pale following contraction of the blood vessels. When a frozen part is warmed it becomes hyperæmic and œdematous, as a result of the dilatation of the vessels. The erythema and anæsthesia may persist for some time, rarely longer than ten

days, the length of time usually depending upon how long the anæmia persisted. An ugly redness of the point of the nose and of the margins of the ears, resulting from a permanent dilatation of the blood vessels, often persists after frostbites of these parts.

(*b*) *Second-Degree Frostbites.*—In frostbites of the second degree the deep red or violet, cold and insensitive skin becomes covered with blebs or vesicles. There is a stasis of blood in the small cutaneous veins, as a result of the contraction of the small arteries, for there is not force enough to drive the blood into the larger veins. Transudation of the blood plasma, resulting in an œdema and the formation of blebs, follows the stasis. When the circulation is reëstablished sensation returns and the blebs dry to form crusts, beneath which healing occurs without scar formation. If the blebs burst and become infected, painful ulcers form which become chronic if an œdema persists.

(*c*) *Third-Degree Frostbite.*—The third degree of frostbite is characterized by gangrene or the formation of an eschar. The tissues die if they are so much damaged that they cannot react when treatment is instituted, and if the large arteries within the frozen area and at its boundaries are closed by thrombi. The frozen part is rigid. The tip of the nose and the margins of the ears may become so brittle that they may be broken off like a piece of glass. When such a part is warmed, only the symptoms of the second-degree frostbite—œdema, cyanosis, and vesicles—are present at first. The part, however, remains cold, blue, and without sensation, as the circulation is not reëstablished. After some days the large shreds of skin and the nails become loosened, and dark brown crusts begin to appear upon the frozen part. Later dry gangrene develops, and a line of demarcation separating the living from the dead forms. Gangrene occurs frequently when treatment is instituted late.

The three degrees of frostbites are often associated when large areas or many different parts are involved. The effects are not the same in different parts, the protected ones presenting the milder changes, the unprotected the severer. The severest cases, associated with freezing of the four extremities and of different parts of the face, are accompanied by general symptoms. An accurate diagnosis as to the degree of the pathological changes cannot be made within the first few hours, as frequently they do not become pronounced until after some days.

The general condition of the patient is not impaired in local freezing proper, unless infection develops from the vesicles, from the necrotic foci, from the ulcers, or from the zone of demarcation.

Sequelæ.—Long-continuing cyanosis and œdema, associated with painful ulcers, crippled fingers and toes, deep defects in the soft tissues, and disfiguring cutaneous scars, are not the only results of frost-

bites. Transitory or permanent paralysis of different nerves, myogenous contractures resulting from a waxy degeneration and destruction of the contractile substance, and proliferation of the intima of the vessels with subsequent nutritional disturbances ending in the formation of ulcers and gangrene are some of the other sequelæ.

Treatment.—In treating frostbites, the temperature should be gradually restored, as in general freezing. The rapid restoration of temperature is very painful and dangerous, as sudden thawing out of frozen tissues causes profound nutritional disturbances.

In the milder degrees of frostbites, rubbing with snow or particles of ice is the common method of relieving the anæmia.

In the severer degrees the dangers of gangrene should be avoided by relieving as quickly as possible the venous stasis. The most important method for this purpose, which was introduced by von Bergmann, is vertical suspension of the frozen member, to which a well-fitting immobilizing splint has been applied. Frequently the swelling subsides rapidly, and the bluish discoloration disappears when this method is employed. When the venous congestion is relieved in this way the arterial blood circulates through the capillaries much more readily, and the circulation is restored much more rapidly than when the venous blood is allowed to stagnate preventing free capillary circulation. Clinical experience has repeatedly demonstrated the value of early suspension in the treatment of frozen extremities. Ritter's view that venous congestion has a favorable influence upon the regeneration of tissue does not seem to hold good in these cases. Venous congestion, however, even when artificially induced, is of no value when stasis with threatened gangrene is already present.

Undoubtedly the artificially induced arterial hyperæmia advised by Ritter is of value in the treatment of long persisting œdema and cyanosis. The hot-air cabinets of Bier and Krause, in which the limb is allowed to remain for one hour, warm baths combined with cold douches, friction obtained by rubbing, and alcohol compresses are of value in the treatment of these chronic changes. The same procedures are to be employed in the treatment of the paralyses, the muscular contractions, and the circulatory disturbances resulting from changes in the vessels.

The frozen part should always be sterilized and dressed aseptically in order to prevent infection. Ruptured vesicles and loosened shreds of epidermis should be removed, preventing in this way the retention of bacteria. Ulcers and denuded areas should be dressed with salve (zinc oxid ointment) to prevent the dressings from becoming attached. The dressing should be changed frequently, as the profuse secretion which is discharged from the ulcers and gangrenous areas collects beneath them.

The same principles should be followed in the treatment of the phlegmons and gangrene as have already been described in the chapters devoted to the subjects. Amputation, which should be made in healthy tissues, should be delayed until a distinct line of demarcation has formed, unless there is a positive indication for earlier operative interference. In dry gangrene of the fingers and toes and some of the other parts, the gangrenous area should be allowed to separate spontaneously. It may be necessary to perform later operation to improve the results when the gangrenous parts are allowed to separate spontaneously.

It is often difficult to treat successfully the red or bluish red discoloration of the nose which frequently persists after milder degrees of freezing of this organ. Riedinger has recommended injections of ergotin in these cases. Repeated needling and injections of small amounts of alcohol may be tried.

Chilblains.—Chilblains (perniones) are chronic inflammatory, usually circumscribed, swellings of the skin which follow a paralysis of the vessels, serous infiltration, and inflammatory proliferation of the skin and subcutaneous tissues. They occur most frequently upon the hands and feet (especially upon the extensor surfaces, on the outer margins of the foot, and about the heel), more rarely upon the face, occasionally upon the penis. Chilblains are caused by repeated frostbites, especially those following the wearing of wet footwear in cold weather and standing for a long time in cold water. They are most common in anæmic individuals, especially in chlorotic girls.

Chilblains develop as painful, itching nodules which are bluish in color and have glistening surfaces. They disappear in summer to recur in the winter, persisting until the chlorosis, which may be a contributing cause, is cured or the occupation is changed.

Vesicles, pustules, and epithelial defects may develop as the result of mechanical influences (rubbing, pressure of a shoe or boot, scratching). These may become transformed into painful ulcers or deep rhagades, and may be followed by suppuration of the chilblain. The cicatricial contraction following these lesions may cause a deformity.

In the treatment of chilblains an attempt should be made to stimulate the circulation of the part involved. Warm baths, alcohol compresses, painting with tincture of iodin, mildly stimulating salves containing mercury or silver nitrate, and the hot-air apparatus are of value. The affected parts must be protected from infection by cleanliness and the use of antiseptic dressings (zinc oxid ointment dressing maintained in position by adhesive plaster). The dressing which is applied should also protect the chilblain from pressure, rubbing, and scratching. One may prevent the development of chilblains by wearing warm clothing, by avoiding tight shoes and gloves, by wearing overshoes, by carefully

drying the feet after washing or when they become wet, and by improving the general condition.

LITERATURE.—*Giese.* Experimentelle Untersuchungen über Erfrierungen. Habilitationsschrift. Jena, 1901.—*Riedinger.* Ergotin gegen erfrorene Nasen. Arch. f. klin. Chir., Bd. 20, p. 457.—*Ritter.* Die Behandlung der Erfrierungen. Deutsche Zeitschr. f. Chir., Bd. 58, p. 172.—*Sonnenburg.* Verbrennungen und Erfrierungen. Deutsche Chir., 1879.

CHAPTER II

BURNS

HIGH degrees of heat acting upon the surface of the body produce pathological changes which are called burns.

Different Causes of Burns.—Burns may be caused by radiating heat (sun rays, open fires), by flames, by red-hot metal and heated solid bodies, by boiling water and hot liquids, by hot steam and gases (especially steam liberated from bursting boilers and gases generated in the explosion of powder, dynamite, and coal damp). The pathological changes caused by caustics are similar to those of the mildest burns.

Different Degrees of Burns.—The different forms of heat do not have the same action; for example, radiating heat never causes more than vesicle formation, while direct contact with a red-hot solid body is almost always followed by eschar formation. The results depend upon the time the heat acts and upon its proximity to the part involved. Three different degrees of burns are recognized. The first-degree burn is characterized by hyperæmia or erythema, the second by the formation of vesicles, the third by eschar formation. The three degrees are often associated. A fourth degree, characterized by charring and disorganization of the burned part, is sometimes recognized.

Burns of the First Degree.—The first-degree burn (combustio erythematosa) is characterized by a reddening of the skin, which is caused by a dilatation of the cutaneous vessels. The redness is accompanied by pain, which increases for a few hours and then gradually subsides, and by some swelling which imparts to the affected part a sensation of tenseness. Sunburn (erythema solare) is the most common and best example of a burn of the first degree. The first-degree burn is always present in the severer burns. It is much more easily produced in children than in adults.

The redness disappears in about two days. It is followed by a brownish discoloration which in turn disappears, when in about five days the fissured horny layer of the epidermis is cast off in shining scales.

If the areas deprived of their horny layer are then again exposed to radiating heat, a second-degree burn characterized by the formation of vesicles is produced, for the areas are very sensitive. The formation of vesicles is then followed by the development of scaling crusts (eczema solare). Sunburn is frequently followed by pigmentation of the skin, causing the brownish discoloration so well known to all.

The burning pain associated with burns of the first degree is usually readily relieved by the application of an ointment (lanolin, vaseline, zinc oxid). These ointments, applied before exposure to heat, will prevent the development of a burn of the first degree.

Burns of the Second Degree.—A burn of the second degree (combustio bullosa) is characterized by the formation of vesicles within the reddened and swollen cutaneous area. The vesicles, varying in size, which develop within a few hours or after a number of days contain a clear serous or light yellowish turbid fluid or a clotted gelatinlike mass. When the vesicle bursts or the cuticle is removed, the cutis vera, red and painful, is exposed. These areas, unless protected, afford infection atria for mild and severe infections.

The contents of the vesicles may be absorbed, and then they later collapse and become replaced by a thin crust. If infection does not occur the crust drops off in a week, a new epithelium having developed beneath it from the stratum Malpighii. In a short time a slight reddening is the only evidence which remains to indicate the location and extent of the burn. A large flat scar remains after a burn of the second degree only when the corium has suppurated.

That severe pain is associated with burns of the second degree is well known to everyone. The pain gradually subsides on the third or fourth day, when the swelling and redness disappear.

The most common and purest type of a burn of the second degree is one caused by scalding with hot liquids or steam or by contact with a naked flame.

The indications in the treatment of a burn of the second degree are to relieve the pain, and to provide conditions which will favor rapid healing. Pain should be relieved by the application of a well-fitting protective dressing; if necessary, by morphin. Healing is more rapid when a dry dressing is employed, provided there is no suppuration, than when ointments, moist dressings, or continuous baths (which macerate the tissues and provide new infection atria) are used.

Since thorough sterilization is impossible because of the pain and the danger of rupturing the vesicles, the grosser particles of dirt should be removed by sponges saturated with alcohol or a three per cent solution of hydrogen peroxid.

All ruptured vesicles and denuded areas should be regarded as in-

fected. The detached shreds and strips of the epidermis should be removed with scissors and forceps, as bacteria become lodged within and beneath them. Vesicles which have not been ruptured should be incised at their bases with a sterile knife, for when they collapse the epidermis forms a protecting covering to the underlying tissues to which it becomes adherent. Small vesicles should not be opened.

After the grosser particles of foreign matter have been removed, a dry dressing or a moist dressing saturated with a boric acid or soda solution, evaporation from which should not be prevented, should be applied. The lower layers of such a dressing become attached to the denuded area. They should be allowed to remain until separated spontaneously, unless there is an indication for earlier removal. The dressing employed by Bardeleben, consisting of gauze, the meshes of which

Fig. 236.—Hypertrophic Scar of the Forearm and Hand following a Burn Received in a Theater Fire.

contain equal parts of powdered starch and bismuth, is also to be recommended. The gauze forms, with the secretion from the denuded area, a protecting crust beneath which healing occurs in from one to two weeks. When the dressing is to be removed the crust should be moistened with vaseline or oil. If an infection occurs, the development of which is indicated by fever and increasing pain in the wound, the dressings should be changed.

Burns of the Third Degree.—The third-degree burn (combustio escharotica) is characterized by the formation of an eschar. The tissues are destroyed to various depths by the direct action of the heat (most commonly by contact with red-hot metal or flames), and a hard, insensitive, sometimes yellow, at other times brown or black mass, the vessels of which are closed by thrombi, forms. This mass, like any other form of necrotic tissue, is separated from the surrounding parts by the de-

velopment of granulation tissue. The eschar following freezing develops slowly, while that following burns develops immediately.

Burns of the first and second degree are usually found in the area surrounding the eschar. During the separation of the necrotic mass mild pyogenic and putrefactive infections, accompanied by local and general symptoms, frequently develop. Occasionally the separation of an eschar involving deep tissues is followed by the opening of joints and body cavities, the erosion of large blood vessels, thrombosis and embolism, and the development of chronic suppuration ending in amyloid degeneration of the viscera.

FIG. 237.—SCAR APPROXIMATING THE CHIN AND CHEST WHICH FOLLOWED THE HEALING OF A SCALD PRODUCED BY BOILING WATER.

When the eschar is cast off a granulating wound, which is inclined to form excessive amounts of scar tissue when contraction occurs, is exposed. The radiating, red and hard, frequently keloidlike scars which follow burns are to be feared, not only because they are disfiguring, but also because they frequently cause adhesions between different parts, thus interfering with the functions of the same. Scars upon the face and neck produce frightful disfigurement (ectropion of the lids and lips and cicatricial adhesions, for example, between the chin and neck). Developing in the groin, in the popliteal space and axillary fossa, about the elbow, and upon the flexor surface of the wrist joint, they may cause contractures. The arms may become immovably attached to the thorax when a burn involving corre-

sponding surfaces of these two parts cicatrizes (Fig. 238). If traction is continually exerted upon a scar, fissures develop which suppurate and

FIG. 238.—CICATRICIAL ADHESIONS BETWEEN THE ARM, THORAX, AND BACK FOLLOWING SCALDING.

lead to the formation of chronic ulcers. Occasionally a carcinoma develops within such an ulcer.

The treatment is the same as described when discussing burns of the second degree. The crust formed by the desiccation of the secretion is the best protection against putrefactive infections. The area may be dressed with bismuth powder until the burns of the second degree are healed. As soon as the granulation tissue begins to secrete pro-

fusely, dry dressing (which should be changed frequently) should be applied.

In extensive burns, after separation of the necrotic tissue, cleansing of the granulation tissue may be hastened by placing the patient in a warm-water bath. The same results may also be obtained by the use of ointments and moist dressings. If the granulating wound appears clean, skin-grafting should be performed at once, in order to reduce to a minimum the amount of cicatricial tissue. If the scar is disfiguring or interferes with function, it is often advisable to excise it and to graft the resulting wound. If the scar is extensive, it may be necessary to perform a number of operations, or merely to excise the part of the scar which causes the greatest functional disturbance. An attempt may be made to soften the scar by injecting thiosinamin, if excision is not desired or is impossible.

Secondary phlegmons, which develop most commonly during the separation of the eschar, should be incised. If they are associated with severe general infection, amputation should be considered.

Carbonization.—Carbonization is observed in cadavers found after fires and mine explosions. Single extremities or parts of the same may be carbonized when molten metal is poured over them. An imbecile or insane person occasionally holds an extremity in the fire until carbonization occurs. The burned part, when carbonized, becomes transformed into a charred, brittle mass which requires amputation.

General Symptoms.—General symptoms are wanting if the area affected is small and there is no infection. If the temperature is carefully observed it will often be found that a general reaction accompanies even the milder burns. The temperature gradually rises, returning to normal in about twelve days, and albumoses, indicating increased destruction of albumins, appear in the urine (Wilms).

If more than one half of the surface of the body is burned, independent of the degree of the burn, severe general symptoms, which almost always terminate fatally within the first few days, may develop. Any burn that involves one third of the surface of the body is serious, and is apt to prove fatal. This fact was first emphasized by Billroth. Even if the individual withstands the immediate shock of an extensive burn and the general symptoms which develop during the first few days, he may die later of infection or pneumonia.

A badly burned patient is at first conscious, but has no idea of the gravity of his condition. He is restless, throws himself about, cries out with severe pain, pleads for relief, and complains of great thirst. The unburned skin is white; the temperature is two or three degrees lower than normal. This reduction in temperature is partly due to the rapid radiation of heat from the capillaries which are exposed in the burned

area, and partly to cardiac weakness. Vomiting, occurring soon after the burn is received, is a bad prognostic sign. In the severest cases the patient becomes apathetic and unconscious in a few hours; the pulse becomes small and rapid; the respirations very rapid and superficial. Vomiting, diarrhœa, cyanosis, delirium, clonic spasms, coma, and collapse, which are more pronounced in some cases than in others, complete the clinical picture, which soon ends in death. The urine is scanty; anuria may be present. Frequently the temperature rises rapidly before death. Because of the rapid radiation of heat from the skin, the rectal temperature may be three or four degrees higher than the axillary temperature.

Pathological Anatomy.—The pathological changes found by post-mortem examination are so slight that they can scarcely account for death. Venous congestion of the viscera and of the cranial sinuses; slight œdema of the brain and its membranes; ecchymoses of the mucous membrane of the gastrointestinal tract, of the muscles, and of the serous membranes; rarely duodenal ulcers following thrombosis and subsequent digestion of the necrotic area, and often slight parenchymatous degeneration of the kidneys are found.

Causes of Death.—Opinions differ widely concerning the cause of death following burns when it is not due to secondary diseases and infection. Sonnenburg believes that death following immediately the reception of a severe burn is due to overheating of the blood. In other cases death is due apparently to a combination of different causes. In some cases death is probably due to the destruction of red corpuscles, and the injury done the kidneys by the excretion of degeneration products; in other cases to the shock following a reflex loss of vascular tone and secondary cardiac paralysis; and in still other cases to extensive capillary thrombosis following the degeneration of blood corpuscles. Recent investigators lay more stress upon the diminution in the amount of blood plasma as the cause of death, especially in burns of the second degree (Wilms), and upon the absorption of toxic substances from the burned area, especially in burns of the third degree. These toxic substances, when absorbed, probably interfere with the metabolism of albumins (Wilms) and cause degeneration and inflammatory changes in the viscera, especially in the brain (Dohrn). It has not been possible, however, to demonstrate toxic substances in the burned tissues (Helsted) or in the blood (Burkhardt). Therefore the solution of the red corpuscles is not to be regarded as due to toxins, but the result of the direct action of the heat (Burkhardt, H. Pfeiffer, Helsted).

Indications for Treatment.—The indications in the treatment of severe burns are: (1) To control pain; (2) to combat shock; (3) to restore the fluids which are being lost by the extravasation of serum;

(4) to aid in the elimination of toxic materials from the body; (5) to prevent infection. If collapse is threatened, subcutaneous or intravenous injections of salt solution should be given. The loss of heat following the destruction of large areas of skin is often followed by collapse. It should be counteracted by placing the patient in a warm bed and enveloping the uninjured parts of the body in cotton and warm cloths. It is often advisable to float the patient in a bathtub filled with warm water.

X-ray Burns.—X-ray burns, resulting from too close, too long, and too frequent exposures, are divided into the acute and chronic. They do not appear immediately after exposures, but become evident somewhat suddenly after a week. In the mild cases there may have been no premonitory symptoms after the exposure, except a slight transitory redness.

In the acute cases the pathological changes correspond to those already described in discussing burns. The mild burns are characterized by erythema, the severe ones by the formation of vesicles, the severest by the destruction of tissue. When the eschar is cast off, the painful X-ray ulcer remains. The mildest changes consist of a falling out of the hair in the area exposed to the rays. All these changes gradually disappear within a number of weeks. The hair grows again after six or eight weeks and the ulcers heal. The skin in which vesicles have formed frequently remains atrophic.

The chronic lesions which not infrequently develop, even when the exposures are made by skilled physicians and technicians, consist of atrophy of the glands of the skin and falling out of the hair, of atrophy of the skin with abnormal pigmentation and fissuring of the nails, and of the development of painful, progressive, chronic ulcers. Other changes which are frequently associated with an obliterating endarteritis may also develop (Mühsam).

The number of X-ray burns have been greatly reduced of late by carefully regulating the time of exposure, and by using lead plates and other devices which protect the parts.

Similar pathological changes have been observed after the use of radium (Halkin).

The same treatment should be employed for these lesions as already advised in discussing burns.

Lightning-stroke.—Lightning may lacerate or burn the part which it strikes. Burns produced by it are often accompanied by severe shock (Sonnenburg).

The shock produced by a lightning-stroke is, as a rule, followed by immediate death caused by a paralysis of the vasomotor and respiratory centers. An individual who is not killed instantly presents the symp-

toms of cerebral concussion. An individual who has suffered a lightning-stroke becomes unconscious and motionless, the pulse is weak and slow or cannot be felt, the respirations are superficial, the extremities are paralyzed, and there is no response to external stimuli. These symptoms may subside within twenty-four hours or more slowly, the convalescence extending over a period of many weeks. Often a feeling of anxiety and unrest and a paralysis of some of the muscles remain. As a rule these disappear completely, but it may be a long time before the condition of the patient may be regarded as normal. People surrounding an individual who is struck by lightning are usually stunned, but the effects are transitory and rapidly disappear.

The lacerating force of lightning is so great that not only may the clothing be torn in shreds, but an entire extremity may be torn from the trunk. Other injuries are often sustained, as the individual may be forcibly thrown to the ground or hurled some distance by the shock.

Besides burns of different degrees, there are also found the points of entrance and exit of the lightning, the course of which may be traced in the skin and deeper tissues. Deep, round eschars, which correspond to the areas burned in the clothing, are often found in the skin. Reddish brown streaks with vinelike branches and markings radiate in various directions through the skin from the point of entrance. The so-called "lightning figures" are caused by burns of the cutis, by laceration of the cutaneous vessels, and coagulation of the extravasated blood. At the point of exit of the lightning the skin is perforated at a number of points, the skin surrounding the perforations being charred and discolored. Burns, often of the third degree, may be found where the clothing is applied closely to the body, or where there is metal (buttons, coins, spectacles), which is melted by the lightning.

In the treatment an attempt should be made to correct the cardiac and respiratory disturbances by cardiac massage and artificial respiration and by administering cardiac stimulants. The paralyses which remain after recovery should be treated by electricity and massage. The burns should be dressed as described above.

Sunstrokes and Heat Strokes.—Sunstrokes and heat strokes, which not infrequently terminate fatally, are usually due to a high elevation of body temperature. *Sunstrokes* are caused by the direct action of the sun's rays upon the body, especially upon the bare head, of individuals who work or sleep in the sun. The prodromata, consisting of severe headache, dizziness, ringing in the ears, and spots before the eyes, are rapidly followed by unconsciousness associated with convulsions. The face is reddened and injected, the skin hot, the pulse rapid and weak. The temperature may rise to 109° F. or higher. Death may occur within a few hours as a result of cardiac paralysis, the patient not having re-

gained consciousness. In less severe cases the patient may gain consciousness after a longer or shorter period and recover completely, if the body temperature is reduced by sponging or by an ice-pack. Post-mortem examination reveals in these cases a hyperæmia of the membranes of the brain and a cerebral œdema.

Heat stroke is due less to overheating of the body by the action of external heat than to an interference with the radiation and conduction of heat produced by metabolism and muscular activity. In the tropics a heat stroke may be caused by an interference in the loss of heat alone, while in temperate climes there is also, as a rule, an increased formation of heat resulting from muscular action (Musehold).

People who succumb most easily and frequently to heat strokes are those who are compelled to perform hard physical labor in humid weather, and who wear clothing which interferes with evaporation from the skin. Heat stroke is very common in soldiers when marching in closed columns in hot weather and in alcoholics.

Profuse sweating, weakness, distress, and great thirst are the prodromata. Severe headache, dizziness, a feeling of anxiety, and vomiting rapidly follow. The speech becomes thick, the sight dim, the patient holds himself erect with difficulty, or staggers until he suddenly falls, and becomes unconscious and motionless. The face is puffy and cyanotic; the pulse is rapid and thready, if palpable. The heart-tones are weak, often irregular; the breathing is superficial and rapid; the skin is dry and hot; the clothing is wet; the temperature is 104° or 105° F.; the reflexes are diminished or lost; the pupils are narrow and scarcely react. Sometimes general clonic spasms, associated with rigidity of the muscles of mastication and of the back, develop.

A majority (sixty-six per cent) of the severe cases die within a few hours of cardiac paralysis. The milder cases recover, but even when convalescence is well established, disturbances of the central nervous system (headache, dizziness, impaired memory, transitory mental confusion) and cardiac weakness remain.

Post-mortem examination reveals a venous congestion similar to that which occurs in asphyxia, systolic contraction of the left ventricle and dilatation and filling of the right, indicating cardiac paralysis. A heat stroke is to be regarded as a carbonic acid intoxication due to exhaustion of the cardiac and respiratory centers following excessive physical effort and interference with the mechanism controlling the loss of heat.

Treatment demands, as in sunstroke, an immediate reduction of body temperature and stimulation of the heart. Artificial respiration and cardiac massage; injections of camphorated oil; rubbing of the skin; the pouring of cold water over the body and the use of ice-packs; the administration of large amounts of water, or, if the patient cannot

swallow, rectal injection of physiological salt solution, are of great value.

The prodromata of a heat stroke are best treated by removing the patient to a cool, shaded spot, by loosening the clothing, and by administering large quantities of water. A heat stroke may be avoided by wearing proper clothing, by drinking freely of small quantities of water, mild tea or coffee, and by the avoidance of alcoholic drinks.

LITERATURE.—*v. Bardeleben.* Ueber Behandlung von Verbrennungen. Deutsche med. Wochenschr., 1892, No. 23.—*Burkhardt.* Ueber Art und Ursache der nach ausgedehnten Verbrennungen auftretenden hämolytischen Erscheinungen. Arch. f. klin. Chir., Bd. 75, 1905, p. 845.—*Dohrn.* Zur pathologischen Anatomie des Frühtodes nach Hautverbrennungen. Deutsche Zeitschr. f. Chir., Bd. 60, 1901.—*Eyff.* Die Hypothesen über den Tod durch Verbrennen und Verbrühen im 19. Jahrhundert. Sammelreferat (1835 bis 1900). Zentralbl. f. d. Grenzgeb., Bd. 4, 1901, p. 428.—*Halkin.* Ueber den Einfluss der Becquerelstrahlen auf die Haut. Arch. f. Dermatol. u. Syphil., Bd. 65.—*A. Hiller.* Der Hitzschlag auf Märschen. Bibliothek von Coler-Schjerning, No. 14, 1902.—*Kienböck.* Zur Pathologie der Hautveränderungen durch Röntgenbestrahlung bei Mensch and Tier. Wien. med. Presse, 1901, No. 18.—*Musehold.* Sonnenstich, Hitzschlag. In Eulenburgs Realenzyklopädie, 3. Aufl., 1899, Bd. 22.—*Mühsam.* Ueber Dermatitis der Hand nach Röntgenbestrahlung. Arch. f. klin. Chir., Bd. 74, 1904.—*Sacconaghi.* Sulle alterazioni anatomo-patologiche degli organi interni in seguito a scottatura. Lo sperimentale, 1901, Nos. 5–6 (Hildebrands Jahresber., 1901, p. 174).—*Sonnenburg.* Verbrennungen und Erfrierungen. Deutsche Chir., 1879.—*Tschmarke.* Ueber Verbrennungen. Deutsche Zeitschrift f. Chir., Bd. 45.—*Wilms.* Studien zur Pathologie der Verbrennungen. Mitteil. aus d. Grenzgeb., Bd. 8, 1901.

IV. GENERAL RESULTS OF INJURIES

CHAPTER I

COLLAPSE, SYNCOPE, SHOCK

Collapse, syncope, and shock will be considered together, as they are closely allied conditions. They follow frequently, although not exclusively, injuries of all sorts, and are characterized by a sudden depression of all the vital forces, which may be transitory ending in recovery or may be fatal. The clinical pictures are very similar, and often they are due to common causes, and therefore it may be readily understood that frequently one passes into the other.

Collapse.—The most prominent symptoms of the condition known as collapse are a sudden giving way of the strength of the individual and a weakening of the heart. There are a number of different causes of collapse, such as severe hæmorrhage occurring in accidents or operations; injuries of the heart, overexertion of the latter in valvular disease, myocarditis, and disease of the cardiac vessels, and when sudden demands are made upon it, as in pulmonary embolism, when one of the larger branches of the pulmonary artery is occluded and the part of the lung supplied by it is thrown out of action; anæmia of the brain, due to embolism, or occurring when an anæmic patient or one recovering from an anæsthetic is placed in an upright position; poisonings (with snake venom, long-continued anæsthesia, with chemical agents) and bacterial intoxications in which, according to Romberg and others, a central paralysis of the vessels is the principal cause of collapse.

Symptoms.—The symptoms are pallor or cyanosis of the face; cold sweat; cold, usually cyanotic, extremities; dilated pupils; small, rapid, scarcely palpable, irregular pulse; mild delirium; superficial breathing (either rapid or slow); after a short time a clouded mentality, or just before death loss of consciousness; vomiting, and a marked fall of temperature (95° to 96° F.) even in febrile disease. These symptoms may develop in rapid succession, death occurring quickly or after slight temporary improvement, if the heart action cannot be maintained.

Treatment.—If collapse occurs the patient should be placed in the head-down posture, which should be maintained. Injections of camphorated oil should be given to stimulate the heart. A hypodermic syringeful of this oil may be given every fifteen minutes until the condition is relieved. In collapse due to hæmorrhage, physiological salt solution should be given subcutaneously or intravenously, depending upon the urgency of the case. Saline transfusions are also very valuable in collapse associated with poisonings and general infections. Artificial heat should be supplied by wrapping the patient in warm blankets or by placing hot-water bottles about him. Whisky may be administered by mouth if the patient is conscious and can swallow, or may be given by rectum with hot coffee. [Crile has made some important observations on resuscitation when collapse from any cause has reached the stage of suspended animation; that is, when the circulation and respiration have ceased. He describes the method in Keen's "System of Surgery," Vol. I, p. 945, as follows: "The patient is placed in the horizontal or head-down position. The tongue is held well forward, and rapid, rhythmic pressure is applied upon the chest over the heart, thereby providing sufficient artificial circulation during the insertion of the infusion canula directed toward the heart into the peripheral artery or vein. As soon as the salt solution begins to flow into the vessel, thrust a needle of a hypodermic syringe filled with adrenalin chlorid (1:1,000) through the rubber tube near the canula, and during about one minute inject from 10 to 30 minims. In suitable cases the heart will promptly begin vigorous beating, and after some time spontaneous respiration will be established. The circulation should be closely observed, and should there be evidence of failure, a continuous intravenous infusion of a 1:20,000 solution of adrenalin chlorid in normal solution should be given as long as required, at the rate of 2 to 3 c.c. per minute. In the mean time the extremities and the abdomen should be firmly bandaged over plain cotton, or the rubber pneumatic suit should be applied."]

Syncope.—Syncope is a sudden, usually transitory, loss of consciousness due to reflex anæmia of the brain caused by psychical influences. It occurs most frequently in nervous, irritable women and in men addicted to alcohol. The sight of blood or surgical instruments, the change of dressings, severe fright, or the view of an injured person may be the cause which produces the reflex paralysis causing the anæmia of the brain and the symptoms associated with it.

Symptoms.—Cadaveric paleness, nausea, cold sweat, dizziness, and darkening of the field of vision are the prodromata of syncope. With fixed stare and widely dilated pupils the patient sinks, having lost consciousness rapidly and completely, and remains motionless and insensible.

The pulse is rapid and small but regular, the respiration slowed and superficial. As a rule, the symptoms rapidly disappear, a fatal termination being extremely rare. Fatal syncope has been most often observed in greatly excited patients shortly before surgical operations or during operations undertaken under local anæsthesia.

Consciousness usually rapidly returns when the patient is placed in a head-down position, thus counteracting the cerebral anæmia, and when the clothes are loosened. The horizontal position should be maintained for some time after the patient recovers, and small amounts of whisky or wine may be given to advantage. If the patient remains in a deep faint for some time, the same treatment as described under collapse should be instituted.

Shock.—Shock is a condition closely related to collapse in which there is a reflex depression of all the vital forces.

Theories of Shock.—According to many authors (von Leyden, Gröningen, and others), shock is due to an exhaustion or inhibition of the centers in the medulla and spinal cord following excessive irritation transmitted from the periphery. This inhibition or exhaustion of the centers is followed by a depression of the functions of the heart and lungs, the vasomotor, sensory, and motor nerves. Schieffer, as the result of animal experimentation, supports this theory, for he found that shock was much less easily produced in animals in which spinal anæsthesia had been induced than in normal ones. According to others, especially H. Fischer, a reflex vasomotor paralysis is the cause of shock. As a result of this vasomotor paralysis, just as in Goltz's tapping experiments, the veins of the splanchnic area become filled with blood and the blood pressure sinks so low that an anæmia of the nervous system develops. Finally, as a result of these circulatory disturbances the heart ceases to beat. Irritation of the sensory, splanchnic, and other sympathetic nerves, and of the branches of the vagus also plays a rôle in the cardiac inhibition, for a transitory inhibition of the heart can be caused by stimulation of the sensory nerves supplying the abdominal viscera.

Shock may be caused by the concussion or contusion of areas abundantly supplied by sensory nerves, as in severe injuries, or by the injury of a special group of nerves. Shock follows not only machine injuries and gunshot wounds of the trunk or extremities, extensive freezing and burns, but also contusions of the thorax, in which the branches of the vagus nerve are stimulated, and subcutaneous injuries of the abdomen, in which the nerves supplying the peritoneum are irritated by the trauma or by the contents of the stomach or intestine when the latter are ruptured. Irritation of the nerves caused by packing aside and handling the intestines in laparotomies, and by excessive traction upon

the spermatic cord during the freeing of a hernial sac may cause shock.

Age, general weakness, cachexia, and anæmia caused by hæmorrhage or disease are predisposing causes.

Symptoms.—The symptoms of shock are rapid loss of strength; depression of cardiac activity, usually associated with a reduction of body temperature; irregular respiration and interference with the spinal functions as indicated by relaxation of the muscles, diminution or loss of reflexes and impairment of sensation, consciousness being retained (Samuel). In the milder forms of shock the skin is pale and cold, the patient is apathetic or restless, movements and reflexes are sluggish, the pulse is small and running rather than slow, and the respirations are slowed. In the severer forms the skin has a cadaveric pallor and is cold; the lips and cheeks are cyanotic, due to the accumulation of blood in the veins; cold sweat covers the body; the pupils are dilated and react sluggishly, and the patient has a fixed stare; he may belch, hiccough, or vomit; the pulse is slow or but slightly faster than normal, and is scarcely palpable; the respirations are superficial and slow, interrupted by deep inspirations; the body temperature is lowered; the acuity of perception is reduced; the reflexes are sluggish; and the fæces and urine are discharged involuntarily. The mind is always clear, but cerebration may be somewhat slow as the result of the cerebral anæmia.

Torpid and Erethistic Shock.—The condition of apathy in which the patient lies prostrate and perfectly relaxed may be followed by a stage of excitement or anxiety in which the limbs are tossed about and the patient rolls about in bed, attempting to rise. Therefore a torpid shock is differentiated from an erethistic shock. During the stage of excitement the pulse becomes small and rapid, the respiration very rapid, and the face reddened. Frequently torpid passes into erethistic shock; at times merely transitory periods of restlessness occur.

Psychical Shock.—A condition which is caused by some psychical excitement, the symptoms of which are usually transitory but may terminate fatally as a result of inhibition of the heart is called psychical shock. This form is most often produced by sudden fright, the sudden announcement of good or bad news, by unexpected noises (e. g., when a gun is fired near by without warning). In psychical shock consciousness is retained, differing in this way from syncope.

Shock, even in mild cases, not infrequently terminates fatally. The fatal termination, as a rule, is not due to the interference with the function of the central nervous system alone, but is due especially to the loss of blood. If the patient is going to recover the symptoms generally subside within a few hours.

Nothing is more difficult than to determine when the symptoms of shock are subsiding in a patient severely injured, especially if the symptoms of shock are combined, as is often the case, with those of internal hæmorrhage or beginning inflammation (e. g., peritonitis). A rise of temperature is generally associated with infections, and the pulse, which was slow or normal, becomes rapid in both hæmorrhage and infection. Severe collapse resembles shock very closely, and if it develops immediately after an injury it can hardly be differentiated from shock. The differentiation is more easily made when there is some cause for the collapse, such as pulmonary embolism, febrile disease, etc.

Pathologic Physiology of Shock.—The experimental work done by Crile has given us a very clear idea of the pathologic physiology of shock, and also the methods of preventing and treating shock. [In Keen's "Surgery," Vol. I, p. 926, he writes: "An abnormally low blood pressure is the essential phenomenon of the state commonly designated surgical shock. There are many other physiologic changes accompanying shock, but these may for practical purposes be regarded either as results of low blood pressure or as factors of minor importance. Among these secondary factors or results are alteration in respiration and cardiac action, modified mental state, loss of power of both the voluntary and involuntary muscular systems, diminution of the secretion of urine, and lowering of body temperature. So long as blood pressure and circulation are sufficient for normal physiologic purposes, a serious state of shock, despite the presence of any other phenomena, cannot be presumed. As Mummery has pointed out, the fall in temperature in shock is largely a result and not the cause of the low blood pressure. The exact physical state of the vasomotor center during the existence of shock has not as yet been satisfactorily demonstrated. The result of that physical state is an inactivity of the center or centers, thus causing a low blood pressure. But it is not certain that we can at present state just what exhaustion or paralysis is. In a certain sense the vasomotor center in a state of shock may be designated as paralyzed, or in a certain other sense as exhausted. We do know, however, that whatever the exact physical state is, one may conclude from the physiologic test of complete recovery that the centers are not physically damaged, that the impairment or breakdown is functional and temporary. It is at once apparent that in the management of operations prevention is more important than treatment."]

[The accompanying blood-pressure tracing indicates the sudden fall in blood pressure obtained by irritation of the inflamed pleura by a trocar. The sudden deaths associated with aspiration, which are not infrequent, are apparently due to shock induced by stimulation of the filaments of the pneumogastric and sympathetic nerves supplying the

pleura, leading to a reflex paralysis of the vasomotor centers in the medulla oblongata and spinal cord (Capps and Lewis).]

Prognosis.—A marked lowering of the temperature and a continued depression of cardiac function, even when powerful cardiac stimulants are administered, are bad prognostic signs.

Treatment.—The indications in the treatment of shock are to prevent further irritation, especially pain, and to improve the cardiac function and circulation. Painful manipulation should therefore not be attempted, and friction of the skin and the application of mustard plasters are to be avoided. Just as in collapse, attempts should be made to aid the circulation by placing the patient in the head-down position, to stimulate the heart by injections of camphorated oil and heart massage, to aid breathing by artificial respirations, and to supply heat by hot-water bottles or bags. Whisky in hot coffee and hot extract of beef given by mouth or rectum have a favorable influence.

FIG. 239.—TRACING INDICATING THE FALL IN BLOOD PRESSURE ASSOCIATED WITH SHOCK INDUCED IN ANIMALS BY IRRITATION OF AN INFLAMED PLEURA AND THE EFFECT OF ADRENALIN INJECTED INTRAVENOUSLY. Capps and Lewis.

If erethistic shock has developed, the patient should be quieted by a hypnotic, preferably by morphin. As these cases are apt to be accompanied by fever, there is no necessity of supplying external heat.

[Crile summarizes the treatment that should be employed in shock as follows: "Physiologic rest is the most important consideration in the treatment of shock. The patient should be kept at rest mentally and physically. Surgeons and nurses should bring assurance and confidence. The patient should be made comfortable. If this cannot be satisfactorily accomplished by management and nurs-

ing, then give a minimum of anodynes. It is not well to tax the patient with unimportant annoying routine measures. The foot of the bed should be elevated. In more critical cases the extremities and the abdomen may be snugly bandaged. Saline solution per rectum, subcutaneously or intravenously, according to the urgency, may be given.

" If the foregoing seems unavailing, 15 minims of adrenalin chlorid (1:1,000) may be added to 500 c.c. saline solution, which is administered subcutaneously, and in extreme urgency a continuous infusion of 1:20,000 adrenalin solution at the rate of 2 c.c. per minute should be tried."]

Anæsthesia and any operative procedure are to be postponed if the patient does not react to the treatment above outlined. Life-saving operations, such as ligation of large vessels, laparotomies, and tracheotomies must be performed even when there is shock.

LITERATURE.—*H. Fischer.* Ueber den Shock. v. Volkmanns Samml. klin. Vortr., No. 10, 1870.—*Gröningen.* Ueber den Shock. Wiesbaden, 1885.—*Georg Hirsch.* Ueber den Shock. I.-D. Halle, 1901.—*Krehl.* Patholog. Physiologie. Leipzig, 1904.—*De Quervain.* Shock. Kochers chir. Enzyklopädie, 1903.—*Samuel.* Shock. Eulenburgs Realenzyklopädie, 1889, Bd. 18.

CHAPTER II

DELIRIUM TREMENS

DELIRIUM TREMENS develops not infrequently in drinkers, most commonly in whisky drinkers, less often in wine and beer drinkers, after injuries, especially after fractures and operations. The greatest number of cases naturally occur in men, as they are more often addicted to drink than women. It is most common in the fourth and fifth decennia.

Prodromata and Symptoms.—The prodromata, which usually develop on the day following the injury, consist of restlessness, sleeplessness, a fine tremor of the hands and tongue, and great irritability. The patient talks to himself, throws himself about in the bed, and picks at his dressings or pulls upon the restraining bands or sheet. After a few hours hallucinations develop, and he sees animals of all kinds in the room and bed. He feels them upon his body and attempts to pick them off and drive them from the bed and room. In this sleepless condition he is still more disturbed by imagining that the air is smoky and that it is choking him, that the room is a prison and that the attendants are guards. The condition often rapidly becomes worse, and the patient

becomes violent as delusions of persecution develop. The delirious patient shouts like a maniac, pours out invective, and strikes at the attendants, particularly when efforts are made to restrain him or place him in bed. There is no sense of pain, and he uses an injured or operated extremity as a normal one; for example, he may walk upon the stump of a recently amputated leg, or beat upon the wall or bed with a severely inflamed arm.

Cardiac Weakness Occurring in Delirium Tremens.—The greatest danger is cardiac weakness. During the stage of excitement the heart may suddenly become weak and rapid or stop, and the patient passes into collapse, which may end fatally. The heart muscle in nearly all these patients has undergone fatty changes as the result of long-continued alcoholism, and when extra work is put upon it during the stage of excitement it becomes rapidly exhausted. In the favorable cases, which are the most common, the patient falls into a long, deep sleep, after the delirium has lasted for some days. When he awakes his mind is clear and he has no recollections of the preceding hallucinations. In the less favorable cases the symptoms gradually subside, but fatal collapse may occur at any time in these cases.

The milder cases take an unfavorable turn if an infection or pneumonia develops or a hæmorrhage occurs, as the weakened heart is then not able to perform the extra amount of work demanded of it.

Prophylaxis.—An attempt should be made to prevent the development of delirium in patients addicted to alcohol, and for this reason alcoholic drinks should not be withdrawn at once, but during the first week following the injury or operation the usual amounts of whisky, cognac, port wine, or other alcoholic drinks should be given. Even after the development of the prodromata the severer stages of the delirium can often be prevented if some alcoholic drink is administered. According to Bonhoeffer, alcohol should be completely withdrawn after the symptoms have developed, for it has no influence upon the later course of the disease.

Essential Cause.—Nothing definite is known concerning the essential cause of delirium tremens. According to later investigations trauma is not so important a factor as the exciting cause as are infections (particularly pneumonia) and diseases of the respiratory passages (fat embolism following fractures). The sudden withdrawal of alcohol is regarded by some as an important factor, but others attach much less significance to it.

Treatment.—When the delirium has developed, morphin, chloral, and scopolamine (gr. $\frac{1}{100}$ subcutaneously) should be given to quiet the patient. Injections of camphorated oil (which can rarely be dispensed with) should be given as a heart stimulant. The patient should be fed;

hot milk, to which are added a few drops of tincture of capsicum, being most often given. [It is absolutely essential that the patient receive sufficient nourishment. It has been suggested by some that the delirium which occurs in these patients is closely related to the delirium which develops during starvation. Patients suffering with delirium tremens should never be etherized or chloroformed, as these two drugs have the same physiological action as alcohol, and when the patient recovers from the anæsthesia the symptoms are often aggravated.]

A large dressing, held in place by plaster of Paris, should be applied to protect the wound or wounds from injury and infection. The patient should then be placed in a specially prepared room and watched by one or two powerful attendants. A restraining jacket is required only in the severest cases.

LITERATURE.—*Bonhoeffer.* Zur Pathogenese des Delirium tremens. Berl. klin. Wochenschr., 1901, p. 832.

CHAPTER III

FAT EMBOLISM

AFTER many injuries, particularly after fractures, fat droplets gain access to and circulate in the blood, this being indicated by the presence of fat droplets in the urine.

Symptoms.—Distinct clinical symptoms, which may lead to death, are rare. The symptoms depend upon the amount of fat which gains access to the blood and upon the viscera involved, and not upon the extent of the injury, for a simple fracture of the fibula, malleoli, or patella may be followed by a fatal fat embolism. It is doubtful whether a fracture (with crushing of the marrow) or the crushing of the panniculus adiposus is absolutely essential for the development of fat embolism, for Ribbert has been able to produce fat embolism experimentally in rabbits by percussing the tibia, not, however, by fracturing the bone. He has come to the conclusion that the general concussion to which the bony system is subjected in injuries, and particularly in fractures, is the cause of the liberation of fat, which is then absorbed by the lymphatics and carried into the blood. He does not believe it probable that the fat is absorbed from the crushed bone marrow at the seat of fracture, for the bleeding veins and those which are closed by thrombi are not able to absorb the fat. Fat embolism has also been observed after the forcible correction of contractures (Payr, Ahrens, and others).

Symptoms develop as soon as a large amount of fat reaches the viscera, for the fat occludes the smaller vessels and interferes with the function of the viscera involved. The fat emboli first lodge in the lung and a hæmorrhagic infarct develops, the symptoms of which are rapid respiration, dyspnœa, pallor followed by cyanosis, failure of circulation, and hæmoptysis. If the fat remains in the smaller arteries of the lung and the heart action is not strong enough to drive the foreign mass into the general circulation (Ribbert), the symptoms are limited to the lungs. If, however, the fat is driven through the capillaries into the general circulation, other viscera, especially the brain and heart, become involved. To the symptoms already described will then be added those of severe cerebral disturbances (delirium, convulsions, vomiting, paralysis, and coma), following multiple small hæmorrhages into the brain, and acute cardiac weakness, following degeneration of the heart muscle. Frequently there is some fever, due in some cases to a beginning pneumonia, in other cases to interference with the heat center by hæmorrhages. The hæmorrhages may also cause a fall in temperature (Czerny, Scriba).

In the severe cases death occurs in a few days. The mild cases gradually recover as the fat is absorbed.

Diagnosis.—The diagnosis of fat embolism can be made with a fair degree of certainty if pulmonary or cerebral symptoms develop soon after an injury to bone in a person who just before the injury was in good physical condition, and if fat droplets can be demonstrated in the urine. Pulmonary embolism caused by the setting loose of venous emboli at the seat of fracture develops much later, usually not before the third week following the injury.

Pathology.—It has been found in cases examined shortly after death that the capillaries and smaller vessels were filled with fat droplets.

Treatment.—The condition should be treated symptomatically. The fracture should be carefully immobilized to prevent more fat from entering the circulation, and remedies should be administered to sustain the heart's action, camphorated oil, ammonia, and digitalis probably being the best drugs for this purpose.

LITERATURE.—*Ahrens.* Tödliche Fettembolien nach gewaltsamer Streckung beider Kniegelenke. Beitr. z. klin. Chir., Bd. 14, 1895, p. 235.—*v. Bergmann.* Zur Lehre der Fettembolie, Habilitationsschrift, Dorpat, 1863.—*v. Bruns.* Die Lehre vonden Knochenbrüchen. Deutsche Chir., 1886, Fettembolie, p. 477.—*Haemig.* Ueber die Fettembolie des Gehirnes. Beitr. z. klin. Chir., Bd. 27, 1900, p. 333, with Lit.—*Payr.* Zur Kenntnis und Erklärung des fettembolischen Todes nach orthopädischen Eingriffen und Verletzungen. Zeitschr. f. Orthopädie, Bd. 7, 1900, p. 338.—*Preindelsberger.* Ein Fall von Fettembolie nach Redressement. Zeitschr. f. Heilkunde, Bd. 24, 1903.—*Ribbert.* Zur Fettembolie. Deutsche med. Wochenschr., 1900, p. 419.

CHAPTER IV

TRAUMATIC DIABETES

It has been shown by the physiological experiments of Claude Bernard that injury of a definite area in the floor of the lower part of the fourth ventricle, between the points of origin of the vagus and auditory nerves, is followed in a few hours by glycosuria if the center controlling the vasomotor nerves of the liver is injured, by polyuria if that controlling the vasomotor nerves of the kidney is affected.

Disturbances of sugar metabolism and of the mechanism controlling the excretion of urine are not infrequently observed after injuries (especially after injuries of the head, after fractures, and injuries of the pancreas, liver, kidney). As a rule, these disturbances develop a few days after the injury. It cannot be demonstrated, however, that there is any lesion of the centers above mentioned. The glycosuria is transitory, subsiding usually within a week. The urine which is secreted never contains more than one per cent of sugar. A true diabetes mellitus or insipidus occasionally develops after an injury. They are much rarer than the temporary glycosuria above mentioned.

Nothing definite is known concerning the nature and cause of traumatic glycosuria.

The treatment is conducted along the lines prescribed in internal medicine.

Literature. *Kausch.* Beiträge zum Diabetes in der Chirurgie. Chir.-Kongr. Verhandl., 1904, II, p. 650.—*Morris.* Diabetes in Surgery. Medical News, 1901, June, 29.

PART V

IMPORTANT SURGICAL DISEASES, EXCLUDING INFECTIONS AND TUMORS

CHAPTER I

DISEASES OF THE SKIN AND MUCOUS MEMBRANES

(a) CONGENITAL SKIN DEFECTS

Congenital defects of the skin appear in a number of different forms, usually associated with disturbances in development. Besides fissures and fistulæ resulting from incomplete fusion of embryonal clefts, there are also adhesions between different parts of the body which are also to be regarded as developmental defects.

Cutaneous syndactylism, in which neighboring fingers are contained in a common cutaneous envelope or are connected by a membrane resembling a web of a goose foot, belongs to the latter class of anomalies. Broad cutaneous adhesions resembling a wing occur in the popliteal space, in the axillary fossa, and between the neck and chest. These malformations are frequently associated with muscular anomalies and other developmental defects.

Small nodules about the size of a pea, occurring in the skin at birth, may be the remains of adhesions between the area in which the nodule appears and the amnion. Deep furrows in the face and upon the extremities, which may even extend down to the bone, may be caused by the constriction of amniotic bands. These may be circular and extend deep enough to amputate the extremity.

Plastic operations of different sorts are often required to repair the fissures and to overcome the adhesions. Abnormal appendages of the skin and fistulæ should be excised.

(b) ECZEMA

The most superficial inflammations of the skin are grouped under the term eczema. They are of interest to the surgeon in a number of

different ways. An eczema interferes with wound repair. In some cases one is compelled to operate upon skin, the seat of an eczema (e. g., an intertrigo of the skin covering a strangulated hernia), and occasionally the eczema develops later when the skin is bathed with secretion from a deep infected wound in bone or when it is exposed to the action of iodoform. An eczema caused by sublimate solution occasionally develops upon the surgeon's hand which incapacitates him for some time, as the vesicles and pustules developing upon the moist, scaling, and fissured area render asepsis impossible, even when the greatest precautions are taken. The diseased area may also furnish the infection atrium for pyogenic infections (lymphangitis, thrombophlebitis, etc.).

Causes of Eczema.—The causes of eczema are external and internal. Mechanical irritation by rubbing of opposed sweating surfaces (intertrigo or chafing of the scrotum and thigh, in the groin and axillary fossa, and beneath large dependent breasts), or by scratching in scabies, urticaria, prurigo, and insect bites; chemical irritation, especially by agents used for sterilization and in susceptible patients by iodoform; and thermal changes produced by radiating heat or dry cold are the external causes.

Eczema may appear as a symptom in a number of conditions, such as icterus, diabetes, nephritis, chlorosis, and dysmenorrhœa. These may be regarded as some of the internal causes.

Different Forms.—Eczema appears in a number of different forms, one frequently passing into another. The skin itches severely and becomes red (eczema erythematosum), and small red nodules which never become larger than a pinhead (eczema papulosum) may then develop. Vesicles may develop from these nodules (eczema vesiculosum), and if the contents of these vesicles become purulent a pustular eczema (eczema pustulosum) develops. If the epidermis is lost as the result of long-continued irritation or maceration a weeping surface remains, weeping eczema (eczema madidans). When the serum dries the area becomes covered with crusts, or, if pus develops beneath the crusts, with pustules resembling those seen in impetigo. [" It is important to remember that an attack of acute eczema, like other acute diseases, may subside spontaneously, and that this is not less likely to happen because the eruption is extensive. The eruption may become abortive in the first stage (when it resembles a papular erythema) and end with desquamation, or after exudation has taken place. This may gradually become less and dry up, when, after a few exfoliations, the skin becomes sound. The possibility of spontaneous subsidence should always be kept in mind, and be a warning against too energetic treatment. But unfortunately, in accordance with the observed proclivities of eczema, it more often happens that the acute passes into a chronic inflammation, which requires the treatment appro-

priate for that form.''—T. C. Allbutt, '' System of Medicine,'' Vol. IX, p. 508.]

Most Common Sites for Development.—Eczema develops most frequently upon the face, head, neck, hands and feet, the external genitalia, in the axillary fold, and in fat people in all the deep cutaneous folds. Eczema also develops frequently in poorly nourished parts, in paralyzed parts, over large tumors, and in skin the seat of chronic œdema and elephantiasis.

Treatment.—The treatment which should be instituted depends upon the cause. The cause should be removed and the affected area should be protected from injuries, among which rubbing and scratching provoked by the severe itching are the most dangerous. The milder acute forms are usually controlled by a generous application of boric acid or zinc oxid ointment, which should be covered with enough dressings to protect the area. In the treatment of scaling chronic eczemas, it is generally best to employ preparations of tar. The eczema developing upon a surgeon's hands, which usually can be prevented, is readily controlled by the use of a ten per cent zinc vaseline ointment and by wearing gloves until the lesions are healed.

(c) ŒDEMA OF THE SKIN AND MUCOUS MEMBRANES

ŒDEMA OF THE SKIN

The fluids found in the tissue spaces, which are filtered from the blood by the secretory activity of the cells of the capillary walls, and are again taken up by the lymphatic vessels, may in certain diseased conditions collect in the tissues and body cavities. If the fluid collects in the cavities, one speaks of a hydrops articularis, a hydrothorax, a hydropericardium, or an ascites, depending upon the cavity involved. If the viscera, the skin, or mucous membrane are saturated with these fluids one speaks of an *œdema*, or if a large part of the surface of the body is involved of an *anasarca*.

Causes of Œdema.—*Venous stasis* and *disturbed capillary secretion* are the most important causes of œdema. *Lymph stasis* is a much rarer cause.

In venous stasis there is an increased formation of lymph, as the result of increased pressure in the capillaries. If the venous stasis continues for some time, nutritional disturbances develop, the tissues lose their tone, and then the normal movements of the lymph are interfered with and the capillary walls become more permeable.

Œdema follows occlusion of the large veins of the trunk and extremities, unless the occlusion occurs slowly enough to permit of the development of a collateral circulation. The œdema which develops

after the ligation of veins is apt to be transitory. After occlusion by a thrombus, or by the pressure of a tumor, the œdema is apt to be more permanent as the openings of the veins entering into the collateral circulation are apt to be occluded. A transitory œdema frequently develops in an extremity which has been immobilized for some time, when attempts are made to use it again. This is due to the impairment of venous circulation, following a temporary atrophy of the muscles.

Disturbance of capillary secretion is the cause of a number of different forms of œdema. The alterations in capillary secretion are either the results of nutritional disturbances, or of toxic, chemical, thermal, or traumatic injuries to the endothelium of the capillaries. As a result of such an injury the vessel wall acts mechanically like a filter. The less the tension of the tissues, which is lowered by the same causes that injured the vessels, and the higher the blood pressure, the more rapidly the fluids are poured out.

Clinical Forms.—*Inflammatory œdema* accompanying inflammation of bacterial, toxic, and mechanical origin develops in different degrees in the skin, mucous membranes, and viscera.

Hydræmic, cachectic, or *marantic œdema* occurs in those diseases characterized by hydræmia. The hydræmia may be due to a decrease in the albumen content of the blood (in anæmia, cachexia associated with chronic infectious diseases and malignant tumors) or to an increase of water in the blood (nephritis and cardiac insufficiency). Neuropathic œdema occurs occasionally in hysteria.

Myxœdema belongs to the chemical œdemas. It develops when the secretory activity of the thyroid gland is greatly diminished or after its complete removal. The changes are most marked in the skin of the face and extremities. The fluid, which is deposited in the subcutaneous tissues and gives it the tense, somewhat doughy feel, resembles mucin; therefore the term myxœdema. This condition may be relieved by the feeding of thyroid extract (thyroid tablets) prepared from the thyroid glands of calves or sheep.

Œdema due to lymph stasis develops only after the occlusion of the large lymphatic ducts of the extremities or of the thoracic duct. Lymph œdema of the lower extremity and scrotum may follow the extirpation of inflamed inguinal lymph nodes, the collateral vessels being closed by thrombi. Occlusion of the thoracic duct is usually caused by neoplasms.

Œdema ex vacuo occurs principally in the cranial cavity and spinal canal. It develops in all cases in which a portion of the brain and spinal cord is lost and its place is not taken by some other tissue.

Appearance of Œdematous Area.—In œdema of the skin the subcutaneous tissues are also involved, for their anatomical structure permits of the accumulation of large amounts of fluid. Not infrequently the

fluid extends from subcutaneous into the loose intermuscular tissues. An œdematous area is swollen, the swelling gradually being lost in the surrounding healthy tissues, and the skin is cool and of a waxy appearance. The skin has a bluish color only when there is a venous stasis. The movements of an œdematous extremity are restricted. It feels heavy and becomes easily exhausted when movements are made. If an incision is made in an œdematous part a clear fluid pours out from the cut surface, the conditions being very similar to those found in an area when infiltration anæsthesia has been employed.

Development of Œdema.—An œdema of the skin never develops suddenly. The fluid collects gradually and the swelling develops slowly. The time required for the swelling to reach its maximum development naturally varies in the different cases. The œdema is not permanent if the cause can be removed or its action is only transitory. Chronic œdema leads to trophic disturbances in the skin, and the latter becomes rough and fissured. As the result of the stimulation and proliferation of the subcutaneous tissues a pachydermia may follow a chronic œdema.

Differential Diagnosis.—An œdema can usually be easily differentiated from other somewhat similar conditions, such as the thickening of the skin associated with elephantiasis, diffuse lipomatosis, lymphangiomas, and recent deeply seated hæmatomas. An œdema pits upon pressure, as the fluids are driven out of the meshes of the subcutaneous tissue, and the pit that remains when the pressure is removed disappears slowly, as some time is required for the meshes to fill again. Pitting upon pressure and the slow disappearance of the pit are characteristic of œdema.

Treatment.—In treating an œdema, naturally, the cause should first be removed and the venous circulation should be assisted and improved by elevation of the extremity and by the application of an elastic bandage, exerting mild compression, from the periphery toward the trunk. In hydræmic œdemas the disease to which the œdema is secondary should receive proper treatment and the general condition should be improved. Frequently the latter is impossible, as the œdema is an indication of the beginning of the end. Massage is of advantage in the treatment of all forms of œdema of the skin, excepting those associated with inflammation and thrombosis. A long-continued inflammatory œdema is often benefited by hydrotherapy, which may also be of value in chronic œdema due to other causes.

ŒDEMA OF MUCOUS MEMBRANES

An œdema of mucous membranes is associated with either *inflammation* or *circulatory disturbances.* If it develops acutely as the result of severe inflammation or sudden stasis, the infiltration of the mucosa

and submucosa causes a marked swelling, which in the upper air passage may produce marked disturbances. Œdema of the nasal mucous membranes, rendering nasal breathing impossible, and swelling of the uvula are constant accompaniments of nasopharyngeal catarrh. Œdema of the glottis may be associated with catarrhal and diphtheritic inflammations, submucous phlegmons, and ulcers of laryngeal mucous membrane. It may develop secondary to inflammatory processes adjacent to the larynx and may follow stasis due to the ligation of large vessels (in operations about the larynx). The swelling in this condition may interfere with respiration and threaten the life of the individual.

Surgical Significance of Œdema.—The chronic œdema which is associated with general venous stasis (in heart disease and emphysema of the lungs), or follows occlusion or compression of the veins draining the mucous membrane is, as a rule, of less surgical significance than the chronic inflammatory œdema. The latter leads, especially in the nose and accessory sinuses, to thickenings, and to the development of connective tissue and pedunculated growths, the so-called polyps. Besides mucous membranes stimulated to proliferation by continued irritation afford a favorable base for the development of a number of different forms of tumors, such as papillomas, fibromas, adenomas (in the form of polyps), and carcinomas. Leucoplakia of the mucous membranes of the mouth upon which carcinoma of the tongue, cheeks, and lips frequently develop is apparently caused by chronic irritation (tobacco smoking).

Treatment.—The same methods should be followed in treating inflammatory œdema of the mucous membranes as have been described in discussing acute and chronic inflammations of the same. A marked œdema due to stasis frequently subsides rapidly after multiple small incisions have been made. An œdema of the glottis may threaten life and demand immediate tracheotomy.

(d) ELEPHANTIASIS

The condition known as elephantiasis Arabum—to distinguish it from elephantiasis Græcorum (leprosy)—and as acquired pachydermia is characterized by a thickening of the skin and subcutaneous tissues of different parts of the body. In advanced cases the deeper-lying connective tissues may also be involved. The thickening of the skin and development of connective-tissue masses produce unsightly deformities of the part involved.

Pathology.—Pathologically, two processes—a chronic inflammatory proliferation of the connective tissues and a dilatation of the lymphatic vessels—are combined. Long-continued inflammatory irritation and

local circulatory disturbances are the causes. Thickening, obliteration, and dilatation of the veins and alterations in the skin are also contributing causes.

Clinical Course.—Elephantiasis develops slowly, the clinical course extending over months and years. It, as a rule, develops from a chronic œdema following the radical removal of suppurating lymph nodes or the dilatation or thrombosis of a large number of veins. Elephantiasis due to these causes develops most commonly in the scrotum and lower extremities. Some cases of elephantiasis follow repeated acute inflammations of the skin and subcutaneous tissues, especially erysipelas, lymphangitis, and thrombophlebitis. After each attack the thickening of the skin becomes somewhat more pronounced. These inflammatory processes develop from a chronic eczema, tuberculous and gummatous ulcers, or from fistulæ leading to suppurating bone cavities, and are generally associated with a lymphadenitis. According to Unna, streptococcic inflammations are the ones which most often leave connective-tissue growths. Elephantiasis due to dilatation or obliteration of the veins is most commonly seen in the lower extremities (especially associated with varicose ulcers or the chronic eczema accompanying varicose veins and thrombophlebitis), upon the scrotum, the penis, the female external genitalia, and the face (lips and lids).

FIG. 240.—ELEPHANTIASIS OF THE LEFT LOWER EXTREMITY IN A WOMAN FIFTY YEARS OF AGE. The skin is covered with thick crusts and is traversed by hard, tumorlike masses. Ligation of the femoral artery gave no results.

Clinical Appearance.—The thickening of the skin is sometimes even and symmetrical, while at other times large masses which resemble tumors in appearance and in their independent growth are formed by transverse furrows (*vide* Fibromas). If the skin is not raised to form folds, it remains firmly attached to the subcutaneous tissues. Pigmentation, the formation of scales following increased proliferation of the horny layer of the skin, in many places (especially upon the scrotum) the development of vesicles by dilatation of the superficial lymphatics and lymphorrhœa (so-called lymph scrotum), the formation of crusts following desiccation of the secretions, eczema, fissures, ulcers, and papillomatous growths accompany the connective-tissue proliferation and produce a bizarre clinical picture which may be altered from time to time by the development of acute inflammatory processes (lymphangitis, phlegmons, erysipelas, lymphadenitis). The connective-tissue proliferation gradually involves the muscles, the contractile substance of which becomes atrophic. Finally the soft tissues become converted into a dense, indurated, or soft gelatinouslike mass (elephantiasis dura or molle) which is often traversed by large dilated lymphatic spaces (elephantiasis lymphangiectatica). The size of the bones of the part involved may be increased by periosteal bone formation.

Clinical Forms.—*Endemic Elephantiasis.*—An endemic form of elephantiasis occurs in the tropics (Arabia, Egypt, Hindustan, Central America, etc.). The disease begins insidiously or with acute febrile attacks of lymphangitis. The changes occur in one, more rarely both, lower extremities, in the scrotum and penis, sometimes in the arms, the external female genitalia, the head, and breast. Sometimes rapidly, at other times more slowly, the parts affected attain a size and form scarcely ever seen in sporadic elephantiasis. The scrotum may be transformed into an unshapely, heavy mass, upon the surface of which the drawn-out and invaginated skin of the penis forms a canal leading down to the opening of the urethra. Lymphadenitis and phlegmons developing from a lymphangitis are common during the course of the disease.

Filaria Sanguinis Hominis.—Some of the cases of this form of elephantiasis are caused by infections with the filaria sanguinis hominis (Bancroft). In some other cases the cause is unknown. Filaria sanguinis is a term applied to the larvæ of a worm, which, when sexually mature, is filiform and measures from 8 to 10 cm. in length. The larvæ are 0.35 mm. long and occur in the blood and lymph of man. According to Manson the larvæ are apparently transferred to man by the sting of mosquitoes. They then gain access to and develop in the lymph vessels, producing a lymph stasis associated with inflammation.

Elephantiasis does not always, however, follow infections with filaria sanguinis hominis. The larvæ may be deposited by the blood in the

Fig. 241.—Elephantiasis of the Scrotum. (Native of Java.)

kidneys, causing hæmaturia. Chyluria and chylous diarrhœa follow occlusion of the thoracic duct.

Acquired elephantiasis or pachydermia may be confused with the congenital elephantous forms of lymphangioma, hæmangioma, and fibromas involving nerves (*vide* Tumors, Part VI).

An accurate history as to the previous clinical course enables one to make a positive *diagnosis*. Partial giant growth is easily differentiated, as in the former the bones are also enlarged.

Congenital Thickening of the Skin.—Congenital thickening of the skin and subcutaneous tissues of the extremities following constriction produced by amniotic bands is seen quite frequently upon the extremities. In these cases the bones are not enlarged. The cause of these changes is an interference with venous circulation occurring during gestation.

Rhinophyma.—Rhinophyma, which develops in old people (especially heavy drinkers) and is characterized by an elephantous thickening of the skin of the nose, is a special form of elephantiasis, usually developing upon an acne rosacea (Fig. 242). While dark red macules or soft nodules transversed by widely dilated capillaries develop upon other parts of the face, large red tumorlike masses and lobulated growths, which histologically are characterized by a connective-tissue hyperplasia, form upon the nose. These masses consist of hyperplastic connective tissue, dilated blood vessels, and sebaceous glands which are hypertrophic or have undergone cystic degeneration. It differs from other forms of elephantous new growths, in that the changes are limited to the nose. The hair follicles in and adjacent to such a growth are either undergoing suppurative changes or appear as deep dilated pores, giving to the dark red soft growth a spongelike appearance. There are a number of different views concerning the cause of rhinophyma. Hebra regards the connective-tissue changes as primary, Lassar believes the degeneration of the sebaceous glands and the inflammation of the

FIG. 242.—RHINOPHYMA.

tissue about them to be the essential cause, while Lesser believes that dilatation of the blood vessels gives the stimulus to connective-tissue growth. Kaposi and Dohi regard rhinophyma as an angioneurosis, the circulatory changes causing connective-tissue hyperplasia. In some cases the essential cause is probably congenital.

Rhinophyma has been operated upon with considerable success, part of the mass being removed with a knife, or, because of profuse bleeding, with a thermocautery. Dieffenbach obtained an exceptionally good result in an aggravated case by cuneiform excisions of tissue (Fritze and Reich). If large defects remain after removal of tissue, they may be covered with skin grafts or granulation tissue may be allowed to develop, new epithelium growing from the remaining sebaceous glands to cover it (Rusch).

Treatment.—The condition may be improved if the tumorlike folds and masses are removed. The penis and scrotum, if very large, should be amputated. Fusiform excision of the skin of the face or extremities, when involved, is of great value. The results following ligation of the principal artery (e. g., in elephantiasis of the lower extremity ligation of the femoral or external iliac), which was introduced by Carnochan (1851) and especially recommended by Hueter (1868), are always doubtful. In inoperable cases the circulation should be improved by elevation of the limb, massage, bandages, and adhesive strips. Alcohol injections may also be employed with a view of obliterating the vessels and causing a contraction of the newly formed tissues. The eczema and fissures should receive appropriate treatment. Extensive phlegmons, exhausting inflammatory processes and ulcers which will not heal may render amputation necessary.

LITERATURE.—*Basch.* Ueber sogenannte Flughautbildung beim Menschen. Zeitschr. f. Heilk., Bd. 12, 1891, p. 499.—*v. Bruns.* Ueber das Rhinophyma. Beitr. z. klin. Chir., Bd. 39, 1903, p. 1.—*v. Esmarch* und *Kulenkampff.* Die elephantiastischen Formen. Hamburg, 1885.—*Friedrich.* Pachydermie im Anschlusse an habituelles Gesichtserysipel. Münch. med. Wochenschr., 1897, p. 33.—*Funke.* Pterygium colli. Deutsche Zeitschr. f. Chir., Bd. 63, 1902, p. 162.—*Fritze* und *Reich.* Die plastische Chirurgie. Berlin, 1845.—*Klaussner.* Ueber Missbildungen d. menschl. Gliedmassen. N. F., Wiesbaden, 1905, p. 9.—*Landerer.* Die Gewebsspannung. Leipzig, 1884.—*Lesser.* Lehrbuch der Hautkrankheiten. Leipzig, 1901.—*Manson.* The Filaria Sanguinis and Certain New Forms of Parasitic Diseases. London, 1883;—The Filaria Sanguinis Hominis Major and Minor, etc. Lancet, 1891, p. 4.—*Rusch.* Zur operativen Behandlung des Rhinophyma. Wien. klin. Wochenschr., 1902, p. 333.—*Scheube.* Die Krankheiten der warmen Länder. Jena, 1900.—*Unna.* Histopathologie der Hautkrankheiten. Berlin, 1894.—*v. Winiwarter.* Die chirurgischen Krankheiten der Haut. Deutsche Chir., 1892.

CHAPTER II

DISEASE OF MUSCLES AND TENDONS

(a) CONGENITAL MUSCULAR DEFECTS

A CONGENITAL muscular defect exists if there is an anomalous insertion or complete absence of a muscle (e. g., anomalous insertion of the tibialis anterior or of the extensor digitorum communis). The resulting disturbance in motion is not to be mistaken for paralysis. Malformations, such as syndactylism, polydactylism, or defects in bone may give a clew to the diagnosis. Congenital absence of the trapezius, which has been demonstrated in the congenital high position of the scapula, has been shown to be the cause of this deformity.

(b) ATROPHY OF MUSCLES, SIMPLE AND DEGENERATIVE

Anatomically a simple is distinguished from a degenerative atrophy of muscle fibers. In the former the muscle fibers decrease in size and number, and no other anatomical changes can be demonstrated. In the degenerative atrophy a number of different pathological processes may be combined, such as fragmentation and segmentation of the fibrillæ, fatty degeneration, coagulation, or liquefaction of the myoplasm. The fibrous and fatty tissues later proliferate to replace the degenerated muscle fibers.

Simple atrophy develops most frequently after non-use (atrophy of disuse) of a muscle or group of muscles. It may follow cerebral palsies or accompany as a reflex atrophy injuries and diseases of the joints. In simple atrophy the electrical irritability of the muscle is reduced, but there is no reaction of degeneration.

The results of simple muscular atrophy are a decrease in the size of the muscle, complete or incomplete loss of function, and contractures due to shortening of antagonistic muscles or groups of muscles.

Inactivity and Reflex Muscular Atrophy.—Inactivity and reflex muscular atrophy cannot be sharply separated. It is a well-known fact that muscles decrease in size when patients are bedridden for a long time. It is most pronounced, however, when an extremity is immobilized in splints or a plaster-of-Paris cast for a long period. If there is an injury or an inflammation of the articular end of a bone, or a disease or injury of a joint, a pronounced atrophy of the muscles, especially of the extensors, more rarely of other muscles, develops in one or two weeks. Atrophy of the deltoid, triceps, quadriceps extensor, and gluteal

muscles follows inflammations and injuries of the shoulder, elbow, knee, and hip joints respectively.

The so-called arthritic muscular atrophy is not entirely due to inactivity, as it develops rapidly even when no immobilizing dressing has been applied, and it rarely occurs in hemiplegia, and when it does it is not pronounced. Paget, Vulpian, Charcot, and others believe that muscular atrophy associated with diseases of joints is of a reflex nature. According to the theory advanced by them, irritation is transmitted from the diseased or injured part along the sensory nerves to the cells in the anterior horns of the spinal cord, which have a trophic influence over the muscles related to the joint, and this irritation produces a change in the cells resulting in atrophy of the muscles. Raymond, Deroche, and Hoffa have been able to prevent this atrophy, after producing an arthritis experimentally, by cutting the posterior roots of the spinal nerves, interrupting in this way the reflex arc.

Degenerative atrophy develops in muscles after injuries, inflammation, and circulatory disturbances; during the course of or following infectious diseases (especially typhoid and tetanus, more rarely recurrent fever and general pyogenic infections); in all peripheral paralyses following injury and inflammation of the nerves; and in spinal lesions in which the integrity of the anterior horn cells is destroyed. In the degenerative atrophies following lesions of the cells of the anterior horn or of the peripheral nerves the reaction of degeneration will be present.

The degenerating muscle becomes shrunken and shortened, and marked functional disturbances follow the development of contractures.

Ischæmic Paralysis and Contractures.—Marked interference with the flow of arterial blood is the most dangerous of the circulatory disturbances. The most usual causes are injuries, ligation, embolism or throm-

FIG. 243.—ISCHÆMIC MUSCULAR PARALYSIS AND CONTRACTURE FOLLOWING THE USE OF AN IMPROPERLY APPLIED PLASTER-OF-PARIS DRESSING.

bosis of one of the larger arteries, when a collateral circulation is not established; long-continued action of great degrees of cold; poorly applied, constricting plaster-of-Paris casts; and constriction for purposes of artificial ischæmia maintained for more than two or three hours.

When the circulation is seriously interfered with the muscles become painful, swollen, of a boardlike hardness, and no longer contract. Passive motion is painful and is no longer free. After a few days contractures which become more marked as the involved muscle undergoes cicatricial contraction develop. These contractures may become very pronounced if an entire group of muscles, such as the flexors of the fingers and hand, degenerates. The contractures are less marked, and improvement may follow mechanical treatment if a considerable number of muscle fibers capable of regeneration remain. Von Volkmann was the first to recognize and describe ischæmic palsies and contractures (Leser). According to Hildebrand, the view held up to the present time that the nerves are not involved in this form of palsy is false. Not infrequently sensory disturbances resulting from injuries of the nerves are also present, the latter being injured by the ischæmia or by the pressure exerted by the cicatricial tissue developing in the muscles.

Ischæmic muscular paralysis differs from paralysis of nervous origin in its clinical course and in the absence of the reaction of degeneration.

Course and Prognosis of Different Forms of Atrophy.—The course and prognosis of the different forms of muscular atrophy depend upon the cause, the character, and the degree of the pathological changes. If the cause can be removed, and the degenerative changes are not so extensive as to render regeneration impossible, restoration of function may follow massage, active and passive motion, baths, and, in paralyses following nerve lesions, electricity. Contractures which demand special treatment may develop (*vide* p. 703). In the treatment of ischæmic paralysis, Hildebrand recommends that the nerves be dissected out early from the shrinking, contracting muscles, and be placed beneath the fascia.

(c) THICKENING AND GANGLION OF THE TENDONS

Thickening of the tendons occurs in the form of small nodules or fusiform enlargements upon the flexors of the fingers, and is the most frequent cause of the condition known as trigger-finger. When the patient attempts to open his hand, the finger affected remains flexed when the others are extended, and when the affected finger is extended with the other hand it flies open with a spring or jerk. The finger affected may also remain extended when the other fingers are flexed, and the same spring or jerk occurs when it is flexed with the other hand. There can frequently be felt during these movements a hard, nodular thickening of the tendon which interferes with the free movement of the latter within its sheath. When the tendon sheath becomes sufficiently expanded to allow this enlargement to pass, the fingers become flexed or extended with a jerk.

When the tendon has been exposed for the relief of trigger-finger, the author has often found a limited fibrous thickening upon the surface of the tendon, or a small round nodule resembling a fibroma within a fusiform enlargement; once a small cyst (tendon ganglion), such as has been described by Thorn and Franz.

Frequently small exudations of blood within the tendon cause similar changes and the transitory snapping of all the fingers with the exception of the thumb. This is often observed after long-continued rowing, and is the result of pressure and traction upon the tendons. It often develops on the left hand of recruits, and is due to the pressure of the butt of the rifle.

Ganglia of the tendons, resembling ganglia developing in the capsule of the joint and in tendon sheaths, are probably of traumatic origin, and are to be regarded as degeneration cysts. In rare cases they have been observed in the tendon of the peroneus tertius (Hofmann), in that of the triceps brachii (Borchardt), and in the extensor tendons of the index finger (Morian). They are found most frequently in the tendons of the flexors of the fingers and may be the cause of trigger-finger.

LITERATURE.—*Borchardt.* Ganglienbildung in der Sehne des M. triceps brachii. Arch. f. klin. Chir., Bd. 62, 1900, p. 443.—*Flatau.* Muskelatrophien nach Frakturen, Luxationen und arthritischen Gelenkerkrankungen. Sammelreferat mit Lit. Zentralbl. f. d. Grenzgeb., 1902, No. 8.—*Franz.* Ueber Ganglien der Hohlhand. Arch. f. klin. Chir., Bd. 70, 1903, p. 973.—*Hildebrand.* Ischämische Muskellähmung. Deutsche med. Wochenschrift, 1905. Vereinsbeilage, p. 1577.—*Hoffa.* Die Pathogenese der arthritischen Muskelkrankungen. Chir.-Kongr. Verhandl., 1892, I, p. 93.—*Hofmann.* Ueber Ganglienbildung in der Kontinuität der Sehnen. Zentralbl. f. Chir., 1899, p. 1315.—*Leser.* Untersuchungen über ischämische Muskellähmungen und Muskelkontrakturen. v. Volkmanns Samml. klin. Vortr., 1884, No. 249.—*Lorenz.* Die Muskelerkrankungen. Wien, 1898.—*Morian.* Beitrag zu den intratendinösen Ganglien. Münch. med. Wochenschr., 1900, p. 1766.—*Thorn.* Ueber partielle Zerreissung einer Beugesehne am Vorderarm mit sekundärer Bildung einer ganglionähnlichen Degenerationszyste. Arch. f. klin. Chir., Bd. 58, 1899, p. 918.

CHAPTER III

DISEASES OF THE TENDON SHEATHS AND BURSÆ

Dry Synovitis.—Dry synovitis (synovitis sicca), the counterpart of dry pleurisy (pleuritis sicca), develops in tendon sheaths after overexertion and laceration of the tendons. The terms tendovaginitis and tenalgia crepitans have been applied to this disease, as a grating and creaking sensation is imparted to the palpating finger whenever the inflamed tendon moves. This sensation is caused by a roughening

due to the fibrin which is deposited upon both visceral and parietal layers of the synovial membrane secondary to a small serohæmorrhagic exudate.

The other *symptoms* are severe pain when movements are made, and the development of a long swelling, slightly tender to pressure, corresponding to the position and course of the tendon. The tissues surrounding the tendon also become swollen and infiltrated.

A dry synovitis subsides in a short time; within two weeks even in the severest cases. As the disease is caused by strains and sprains received during work, recurrences are common.

The extensor tendons of the thumbs of laundresses are frequently involved as a result of the wringing of clothes. Not infrequently the extensor tendons of blacksmiths, locksmiths, drummers, and piano players become involved, especially when the work is so heavy or practice is continued so long that the tendons are strained. It is much more apt to develop in novices than in people who are accustomed to their particular lines of work. The tendons of the peroneal and tibial muscles are affected in oarsmen. Similar lesions develop in the tendo Achillis of ballet dancers, although this tendon has no synovial sheath.

The *diagnosis* is easily made. The position and form of the swelling arouses the suspicion of the experienced surgeon at once. The peculiar creaking and grating sensation elicited when the tendon moves to and fro makes the diagnosis positive. This sensation is elicited only when certain movements are made, and only over the course of the tendon; therefore it can easily be differentiated from the crepitus elicited in diseases of the joints and in fractures.

The *treatment* consists of the application of a felt or pasteboard splint to immobilize the tendon involved. The pain disappears in a short time; the other symptoms in a few days. If in severe cases there is still some crepitus at the end of a week, mild massage, active and passive motion are indicated. Excessive use of the tendon or tendons should be avoided for some time.

Serous and Serofibrinous Tendovaginitis.—Serous and serofibrinous exudates frequently follow hæmorrhages into the tendon sheaths, associated with fractures and dislocations. Usually they subside during the treatment of the fracture or dislocation.

Chronic hydrops of the tendon sheaths is, as a rule, of tuberculous origin (*vide* p. 442).

Ganglia of Tendon Sheaths.—Ganglia of the tendon sheaths are not common. They are similar to the ganglia which develop from the capsule of the wrist joint, but usually are smaller than these. Usually they are situated near the metacarpo-phalangeal joint on either side of the sheaths of the flexor tendons. They may exert pressure upon the digi-

tal nerves sufficient to cause a severe neuralgia. The neuralgia rapidly disappears after the cysts are extirpated (Witzel).

Urates may be deposited in the different tendon sheaths and bursæ in gout. Those adjacent to the joint first involved are nearly always affected (*vide* p. 725).

Hygroma.—The hydrops or hygroma is the most common form of chronic inflammation of bursæ. An hygroma follows the incomplete absorption of exudations of blood into bursæ and long-continued mechanical irritation.

The contents of an hygroma are in the beginning thick and mucoid in character, later they become more serous, or, after an injury, hæmorrhagic. The walls of the hygroma are not smooth like those of an acutely inflamed bursa or of one into which blood has been extravasated. They are thickened and contain many recesses, masses, and bands of tissue with wartlike and villous outgrowths (Fig. 244). Often the villous outgrowths are so large and numerous that they fill the cavity of the bursa, which then contains but little fluid, while the thicker and more delicate bands of tissue extend from one to the other wall of the sac. Often the surfaces of these villous masses and bands are covered with old blood clots, which may have become calcified. Free bodies, occasionally a point of a knife or a bullet, may be found in the hygroma. When these foreign bodies are found, they should be regarded as the cause of the chronic inflammation which resulted in the formation of the hygroma.

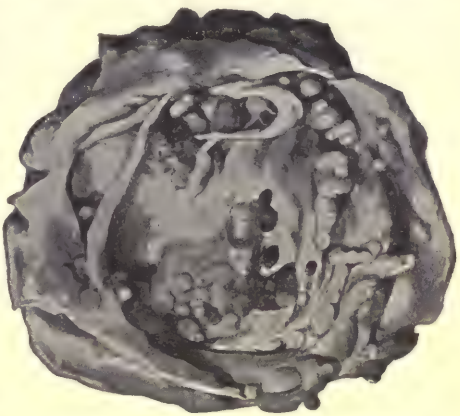


Fig. 244.—Hygroma Bursæ Olecrani.

Chronic inflammation, such as leads to hyperplastic changes in the joints, does not account entirely for the changes in the walls of the bursa above described, although it and the subsequent organization of the fibrinous deposits certainly play an important part. Even in a recent hygroma one finds, as might be expected, not a simple, but a multilocular cavernous cyst (Schuchardt). Some of the masses in the wall of a hygroma and of the free bodies are formed by the cicatricial contraction of the inflamed fatty tissue surrounding the bursa (Graser). According to Langemak, as the inflamed fatty tissue contracts it is transformed into a mass of collagenic scar tissue. After the formation of an interstitial substance which resembles fibrin, this scar tissue lique-

fies, and as a result degeneration cysts form in the wall of the bursa and in the tissues surrounding it. These cysts eventually rupture through the wall of the bursa, and a communication is established with its interior. This accounts for the formation of recesses and evaginations in hygromas. Hygromas are closely related pathologically to ganglia, which are also formed by the liquefaction of tissue (*vide* p. 731).

In the majority of instances an hygroma develops as a painless hemispherical swelling with a smooth surface, covered by normal or slightly thickened skin. In some instances, however, the bursa enlarges rapidly after a trauma when blood is poured out into its cavity and the swelling does not subside. An hygroma may become as large as a hen's egg or an apple; in rare cases even larger. The swelling, sharply defined externally, fuses below with the deeper tissues and is attached, depending upon its position, to the surface of a bone or tendon. It can therefore be displaced but little. As a rule, fluctuation can be elicited when the sac is not too full. Nodular areas can frequently be palpated in the thickened walls of an old hygroma. A peculiar grating or creaking sensation can be elicited upon pressure, even when there is little fluid.

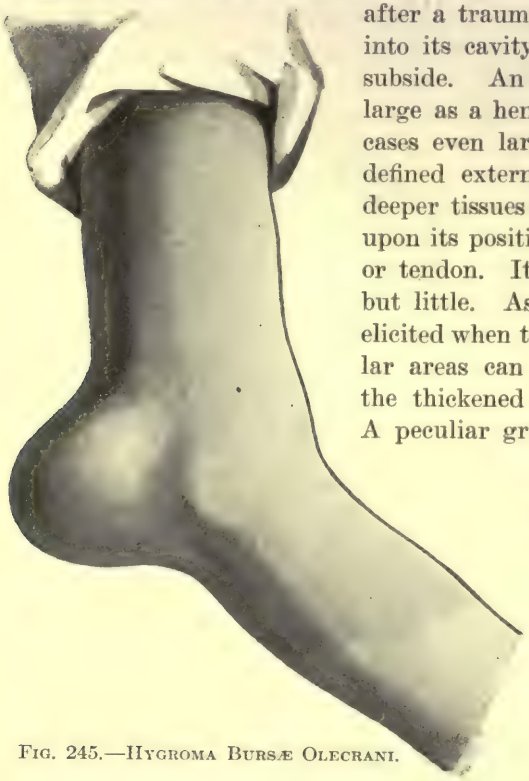


Fig. 245.—Hygroma Bursæ Olecrani.

Most Common Positions for Hygromas.—Hygromas develop upon those parts of the body where a bursa is normally present, or where a bony prominence is continuously exposed to pressure and the development of a bursa is favored. Hygromas of the prepatellar and olecranon bursæ are the most frequent, for these bursæ are not only exposed to injuries, but also to mechanical irritation in a number of different occupations—prepatellar bursa in housemaids and scrub women (housemaid's knee), olecranon bursa in miners (miner's elbow).

Hygromas of the bursæ about the shoulder and hip joints, and popliteal space, follow sprains and dislocations of the joints. Inflammation of the acromial bursa is caused by pressure (being of rather frequent occurrence in hod carriers). The largest hygromas developing in newly

formed bursæ are found on the outer margin of a clubfoot. The hygromas developing in other varieties of bursæ are usually small, the best-known examples being the hygroma developing over the head of the first metatarsal bone in hallux valgus, and over fractures in which there is a subcutaneous angular deformity.

FIG. 246.—HYGROMA BURSÆ PRÆPATELLARIS.

Multiple Hygromas.—The development of multiple hygromas always suggests some infectious cause (gonorrhea, tuberculosis, syphilis), as hygromas of mechanical origin are usually single, or the corresponding bursa on the other side is the only other one involved. Deep-lying hygromas about the hip or in the popliteal space may resemble tumors or gravitation abscesses, and it may be necessary to aspirate the swelling in order to make a positive diagnosis.

Treatment.—The amount of thickening in the wall of the bursa determines the treatment which should be instituted. If the hygroma is recent and the walls are not thickened, aspiration of its contents, followed by the injection of iodin or carbolic acid and compression, may result in a permanent cure. If the walls of the bursa are thickened and indurated, complete excision will probably be necessary. Complete excision is also to be preferred if a fistula is present.

LITERATURE.—*Graser.* Ueber die sogenannte Bursitis proliferans. Zentralbl. f. Chir., 1902, p. 46.—*Heineke.* Die Anatomie und Pathologie der Schleimbeutel und Sehnenscheiden. Erlangen, 1868.—*Langemak.* Die Entstehung der Hygrome. Arch. f. klin. Chir., Bd. 70, 1903, p. 946.—*Schuchardt.* Ueber die Entstehung der subkutanen Hygrome. Chir.-Kongr. Verhandl., 1890, II, p. 1.—*Witzel.* Ganglien an der Greifseite der Hand als Ursache von Neuralgien. Zentralbl. f. Chir., 1888, p. 137.

CHAPTER IV

DISEASES OF THE BLOOD AND LYMPHATIC VESSELS

(a) ARTERIOSCLEROSIS, ATHEROSCLEROSIS

ARTERIOSCLEROSIS (chronic deforming endarteritis, atheroma of the arteries, atherosclerosis of Marchand) may be the cause of a number of important surgical lesions.

The entire process, which is chronic, is of a degenerative nature. It usually begins in adult life, in people of about forty years of age, more rarely in young people. The entire or greater part of the arterial system may be involved in the pathological process. Fatty degeneration and proliferation of the intima, resulting in the formation of foci filled with detritus, ulceration, fibrous induration (sclerosis), and calcification of the vessel wall go hand in hand. A localized form characterized by the formation of small flat nodules, which may undergo fatty degeneration and form atheromatous ulcers (arteriosclerosis circumscripta or nodosa), is frequently combined with a diffuse proliferation of the intima (arteriosclerosis diffusa), which leads to the occlusion of the lumina of the smaller arteries (endarteritis obliterans). Fibrous changes also occur in the tunica media and lime salts are deposited in its muscular fibers. This is often associated with the formation of nodules, especially in the arteries of the lower extremities. The cellular infiltration and thickening of the tunica adventitia in arteriosclerosis is never so marked as in the arterial changes of syphilitic origin (*vide* p. 505).

The arteries affected become hard, irregular, slightly tortuous, and may be easily palpated. Upon section it may be easily seen that the lumina of the arteries are greatly reduced in size. The arteries are

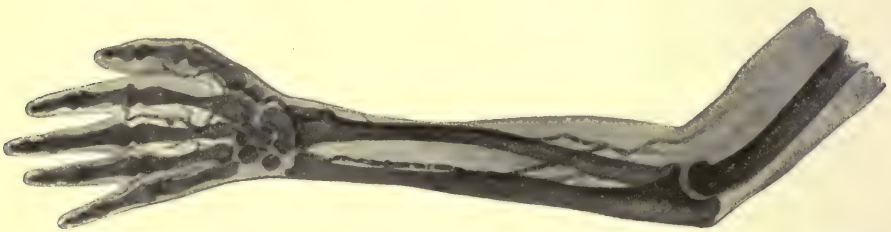


FIG. 247.—SCLEROTIC ARTERIES AS THEY APPEAR IN A ROENTGEN-RAY PICTURE. Male patient, fifty-five years of age, suffering with dry gangrene of the distal phalanx of index finger.

often so hard and fragile that they are ligated with difficulty. When dissected free they appear as yellowish white, irregular, nodular strands; and in Roentgen-ray pictures, if there are enough lime salts, as faint beady shadows, resembling a necklace (*vide* Fig. 247).

Causes of Arteriosclerosis.—There are a number of different views concerning the cause of arteriosclerosis. It may be due to nutritional disturbances in the intima resulting from injury of the media or of the elastic elements of the vessel wall (Marchand), to primary inflammation of the vessel wall (Köster), or to a weakening of the media with compensatory proliferation of the intima (Thoma).

The principal etiological factor is increased intraarterial pressure, which may be continuous or intermittent (Marchand). There are a number of general and local predisposing causes, such as diseases of the central nervous system and peripheral nerves (tabes, syringomyelia, neuritis); the action of a number of different poisons (alcohol, tobacco, ergot, lead, mercury, phosphorus); infectious diseases of all kinds, of the chronic type, especially syphilis and leprosy; constitutional diseases (gout, diabetes); excessive physical effort (with acute dilatation of the arteries); thermal influences (action of mild degrees of cold) (Zoege von Manteuffel).

Results of Arteriosclerosis.—The results of arteriosclerosis are: (1) Disturbances of the general circulation and the function of the different viscera, resulting from the lessened distensibility of the arteries and narrowing of the lumina of the vessels. The interference with the circulation is most marked when the veins are also diseased. (2) Occlusion of the vessels by proliferation of the intima, thrombosis, or embolism, the embolus arising in an atheromatous ulcer in a vessel nearer the heart. If the heart action is bad and the vessels entering into the collateral circulation are diseased, gangrene of the tissues supplied by the artery frequently follows its occlusion (senile gangrene of the toes, embolic gangrene of the extremities). (3) Rupture of the diseased vessel, which is most frequent in the cerebral arteries, causing apoplexy. (4) Aneurysm. (5) Thrombosis.

LITERATURE.—*Fr. Fischer.* Die Erkrankungen der Lymphgefässe, Lymphdrüsen und Blutgefässe. Deutsche Chir., 1901.—*Jores.* Wesen und Entwicklung der Arteriosklerose. Wiesbaden, 1903.—*Marchand.* Arterien. Eulenburgs Realenzyklopädie, 1894;—Die Arteriosklerose (Atherosklerose). 21. Kongress f. innere Medizin. Leipzig, 1904. Verhandl., p. 23.—*Romberg.* Ueber Arteriosklerose. Ibid., p. 60.—*v. Schrötter.* Erkrankungen der Gefässe. Nothnagels spez. Path. u. Ther., Bd. 15, Part 3.—*Thoma.* Die diffuse Arteriosklerose. Virchows Arch., Bd. 104, 1886, p. 209.—*Zoege v. Manteuffel.* Arteriosklerose. Kochers Enzyklopädie, 1901.

(b) ANEURYSMS

By aneurysm (from the Greek ἀνευρύνειν, meaning to widen) is understood a pathological dilatation of an artery. Aneurysms occur in two principal forms, the *simple* or *arterial* and the *arteriovenous.* In the former there is a dilatation of the arterial wall; in the latter a communication between an artery and a vein, with or without an intervening sac. Because of the similarity of clinical symptoms the *pulsating* or *arterial hæmatoma* must be classified with aneurysms, even if the sac is not formed by the wall of an artery, but by blood which has been poured out into the tissues and has coagulated. A *true* is distinguished from a *false* aneurysm. In the former all the tunics of the

vessel are present, while in the latter the sac of the aneurysm contains little or none of the original tunics of the vessel wall. [This distinction, as Cohnheim has said, is artificial and not based upon sound pathological principles.]

The form of an aneurysm differs, depending upon whether the entire circumference of an artery or only a portion of it is involved. If only a portion of the arterial wall becomes dilated a *saccular* aneurysm, if the entire circumference, a *cylindrical* (spindle-shaped, fusiform) or *cirsoid aneurysm* (or better angioma racemosum) develops.

A number of different varieties of true arterial aneurysms have been described, the descriptions being based upon the mode of development.

Congenital Aneurysm.—In rare cases congenital aneurysms of the abdominal aorta (Phaenomenow), of the ductus Botalli (Thoma), and multiple aneurysms of the small arteries, due to congenital defects in the elastic elements of the walls of the vessels, have been observed.

Spontaneous Aneurysm.—Two different forms of spontaneous aneurysm have been described, depending upon the way in which they are produced: *aneurysm by distention* (Thoma), *aneurysm by rupture* (Eppinger). In the former the arteriosclerotic, inflamed, or healing arterial wall is stretched, and the least resistant portion gives way and becomes dilated or evaginated; in the latter the elastic elements of the media and one of the other tunics of the vessel wall are ruptured by a sudden rise in blood pressure, the result of physical effort or mental excitement. The aneurysm by distention is diffuse, sometimes associated with evaginations, and occurs as the fusiform or cylindrical aneurysm. An aneurysm by rupture is, as a rule, a saccular aneurysm. Spontaneous aneurysms develop most frequently in arteries surrounded by loose connective tissues, which afford but little support to the arterial wall.

Aneurysm by Erosion.—If the wall of an artery within an acute or tuberculous abscess becomes inflamed and necrotic, an aneurysm may develop within the necrotic area if immediate rupture does not occur. In these cases the intima often projects through the defect in the media and adventitia, and sometimes these aneurysms are spoken of as *hernial aneurysms.*

Embolic Aneurysms.—An embolic aneurysm develops after the intima has been injured by a hard, sharp embolus which has been set free from a hard, calcareous plaque in an arteriosclerotic artery, or after infection of the intima following lodgment of an embolus containing bacteria (*mycotic aneurysm*).

True Traumatic Aneurysm.—The dilatation occurs in that part of the artery the walls of which have been crushed or lacerated, but not completely destroyed.

Dissecting Aneurysm.—The dissecting aneurysm develops when the intima and media have been ruptured and the adventitia remains intact. [" The blood spreads between the layers of the vessel wall, stripping up the inner from the outer half, the line of cleavage being within the middle coat, half going with the adventitia, half with the intima."—Rose and Carless, " Manual of Surgery," p. 306.] If the blood spreads around the entire circumference of the artery a diffuse cylindrical aneurysm develops; if it is confined to one portion of the arterial wall, a saccular aneurysm forms. The extravasated blood either coagulates in the vessel wall or ruptures through the adventitia.

A **false traumatic aneurysm** develops as a saccular aneurysm from a pulsating or arterial hæmatoma which follows a wound (punctured, contused, gunshot, or lacerated) of the vessel wall. The connective tissue surrounding the hæmatoma becomes thickened to form the sac of the aneurysm which contains none of the tunics of the vessel wall. The blood enclosed within the thickened connective tissue coagulates and surrounds a cavity into which the blood passing out of the opening in the vessel wall flows. The arterial wall proper is not dilated. This variety of aneurysm may develop about the end of an artery in an amputation stump. The small projectiles used in modern rifles produce traumatic aneurysms more frequently than the larger ones formerly used (*vide* Gunshot Wounds).

Age at Which Aneurysms Most Commonly Develop—Vessels Most Commonly Involved.—The simple arterial aneurysm is about twice as frequent in the male as in the female, and develops most frequently between the thirtieth and fiftieth years of life. Aneurysms are most common in the thoracic aorta; then in the popliteal and femoral arteries; appearing next in order of frequency in abdominal aorta, the subclavian, innominate, axillary, iliac, visceral, cerebral, and pulmonary arteries (Crisp). If the small aneurysms are considered, the arteries of the lungs and brain are most frequently involved (Orth).

Character of Aneurysmal Sac.—The sac of a true aneurysm has a thin wall, the tunics of which are altered by the original disease or by the scar in the vessel wall. In the dissecting aneurysm the tunics of the vessel wall are separated from each other by blood, while the small sac of the hernial aneurysm is composed of endothelium alone. The sac of an aneurysm may be thickened by inflammatory processes, and may become closely adherent to surrounding tissues.

The sac of a false aneurysm consists of dense connective tissue. It is adherent to the surrounding tissues and has no endothelial lining. Large false aneurysms contain stratified layers of blood clot which strengthen the wall of the sac and protect it for a long time against rupture. These clots may, however, narrow and occlude the arteries

which anastomose and provide for a collateral circulation or project into the lumen of the artery and be the source of emboli.

Size of Aneurysms.—The size of an aneurysm varies, depending upon the caliber of the vessel involved. The sac of an aortic aneurysm may become as large as a fist.

Enlargement of an Aneurysm.—The growth of an aneurysm is slow, except in the embolic or mycotic variety, and is often retarded or interrupted by thrombus formation and inflammatory thickening of the sac.

Symptoms.—In the beginning the symptoms are not distinct. Even in a false aneurysm, when blood is poured out into the tissues, the symptoms may be obscure or insignificant at first, as the hæmatoma may be deeply situated

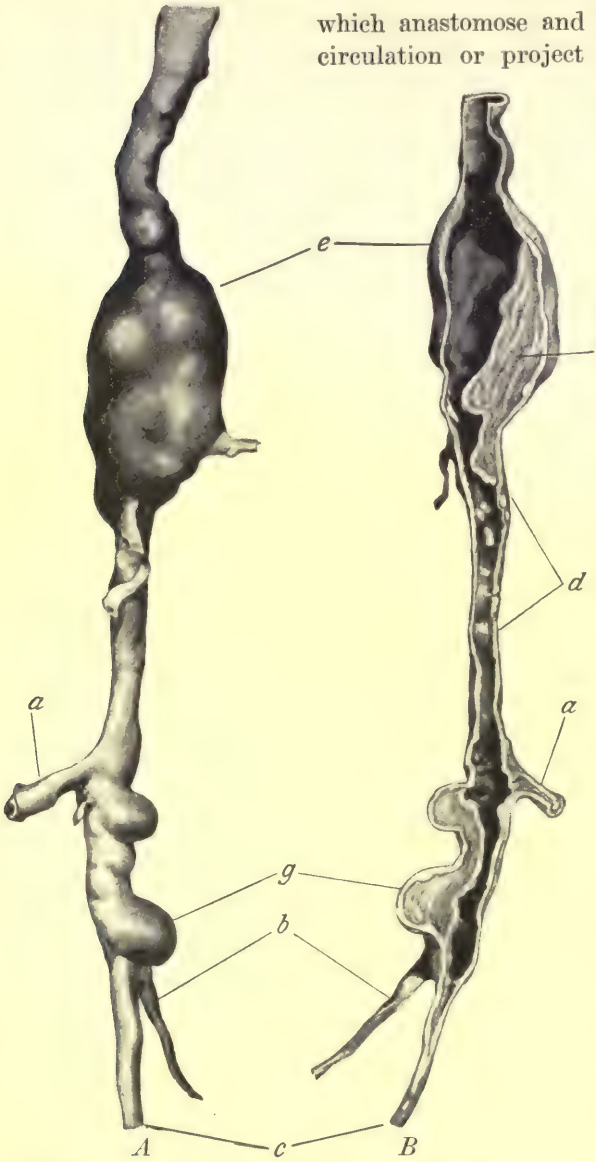


FIG. 248.—*A*, FUSIFORM ANEURYSM OF THE POPLITEAL ARTERY DEVELOPING IN A MALE PATIENT FIFTY-NINE YEARS OF AGE. The thigh was amputated above the knee because of gangrene of the foot. *e*, Advanced arteriosclerosis; *g*, small saccular evaginations in the posterior tibial artery. *B*, LONGITUDINAL SECTION OF THE SAME PREPARATION. *f*, Thick, laminated thrombus in the popliteal aneurysm. The thrombus extends into the posterior tibial artery (*c*) down to the origin of the peroneal artery (*b*), the lumen of which is greatly narrowed. The posterior (*c*) and anterior tibial (*a*) arteries are closed by thrombi. The peroneal artery (*b*) is closed by endarteritis. (*d*) Calcified plaque.

and covered by resistant tissues. The symptoms first become distinct and pronounced when a *visible* and *palpable tumor* develops, or when the tumor exerts pressure upon important structures. *Pulsation* may be seen in the round, oval, rarely sharply defined tumor, if it is superficial. The pulsation is *expansile* in character—i. e., the entire tumor increases in size with each systole and evenly in all directions, so that if the tumor is lightly grasped in any position the fingers will be separated. Pulsation may be transmitted to a tumor or abscess lying near or upon an artery. This pulsation, however, is never expansile, the tumor or abscess being merely lifted with each beat of the artery and not expanding evenly in all directions. Expansile pulsation is therefore of great diagnostic importance. There is felt upon *palpation* of an aneurysm a *soft thrill* or *fremitus*, there is heard upon AUSCULTATION, when the stethoscope is held over the tumor, a *blowing, buzzing bruit*, which is synchronous with systole, but may also be heard in diastole. The latter is produced by whirls in the blood current, formed when the stream entering and leaving the sac meet. The tumor may disappear completely, or almost completely, when pressure is made upon it, to reappear when the pressure is released. The expansile pulsation disappears when pressure is made upon the artery proximal to the tumor. Pressure upon the vessel distal to the aneurysm causes the latter to enlarge.

All these symptoms may be indistinct or absent if there are thick layers of blood clot within the sac.

The *pulse* on the diseased side distal to the aneurysm is *weaker* than on the healthy side and is *slightly delayed*, and the apex of the pulse wave obtained in a sphygmographic tracing is lower and more rounded.

Pressure Symptoms.—Pressure upon adjacent nerves frequently gives rise to severe and distressing symptoms. Unpleasant sensations and pain at the beginning increase to severe neuralgia as the pressure increases. Eventually sensory disturbances and paralyses develop. Pressure upon adjacent veins is indicated by passive hyperæmia, distention of the subcutaneous veins and œdema. The compressed vein may be completely closed by a thrombus.

All these symptoms increase as the aneurysm enlarges. The same symptoms (especially sensory disturbances, numbness, formication, pain) frequently develop immediately after the injury of the artery in traumatic aneurysms, and are caused by the pressure exerted by the hæmatoma. They disappear as the wound heals, but return after a number of weeks, as the sac forms and increases in size.

Clinical Course.—Spontaneous cure of an aneurysm by filling of the sac with a thrombus or thrombosis of the artery proximal and distal to the opening communicating with the sac is rare, and occurs only in the

smaller aneurysms. As a rule an aneurysm, excepting the mycotic variety, undergoes a continued growth, gradually enlarging until it ruptures. A bone adjacent to an aneurysm may be gradually worn away by the pulse beat, as a stone is worn away by water drops. The bone undergoes pressure atrophy. An aneurysm of the thoracic aorta, if it grows forward, gradually destroys the sternum and ribs and reaches the skin. If it grows posteriorly, it destroys the vertebræ and may eventually exert pressure upon the spinal cord or nerves if the sac does not rupture or the patient does not die of some intercurrent infection. The skin covering an aneurysm becomes more and more tense, and finally necrotic, so that eventually the sac of the aneurysm becomes exposed. It may then rupture externally and cause death. If before rupture the poorly nourished skin becomes inflamed, a subcutaneous phlegmon may develop. Rupture of the sac occurs at the point where the blood stream exerts the greatest pressure; that is, where the deposition of layers of blood clot is prevented. Rupture into the pericardial, pleural, and peritoneal cavities is as surely fatal as external rupture and soon causes death, the symptoms being those of internal hæmorrhage. The rupture of a deep aneurysm in an extremity is indicated by the rapid development of a large swelling which soon ruptures externally, as the tissues covering it soon become necrotic as a result of the pressure exerted upon them. A hæmatoma of the neck, resulting from rupture of an aneurysm, may cause suffocation. The rupture of an aneurysm into a vein produces the secondary form of arteriovenous aneurysm.

Complications.—Complications may be caused by separation of particles of a thrombus and embolic closure of the peripheral vessels with subsequent gangrene. Gangrene is more apt to develop when the vessels entering into the collateral circulation are closed by thrombi or are obliterated by arteriosclerosis.

ARTERIOVENOUS ANEURYSM

There are three varieties of arteriovenous aneurysm (Hunter's aneurysm by anastomosis, 1784) (*vide* Fig. 249).

The **arteriovenous aneurysm** with a **venous sac,** the so-called *varix aneurysmaticus* (Scarpa), develops most frequently after the simultaneous injury of the artery and vein at corresponding points. After agglutination of the openings in the vessels the arterial stream passes through the opening in the vein (arteriovenous fistula) and causes a dilatation of the wall of the vein opposite the opening, resulting in the formation of a varix. The spontaneous development of a varix aneurysmaticus is rare. In the cases in which such an aneurysm has developed spontaneously the calcified arterial wall has probably exerted

pressure upon the vein, and later an atheromatous ulcer has extended from the artery through the vein wall.

The varix usually has very thin walls and can be easily shelled out from the surrounding tissues.

The **arteriovenous aneurysm** with a **false sac,** the so-called *aneurysma varicosum* (Scarpa), likewise develops after simultaneous injury of an artery and vein at corresponding points. In this form, however, a

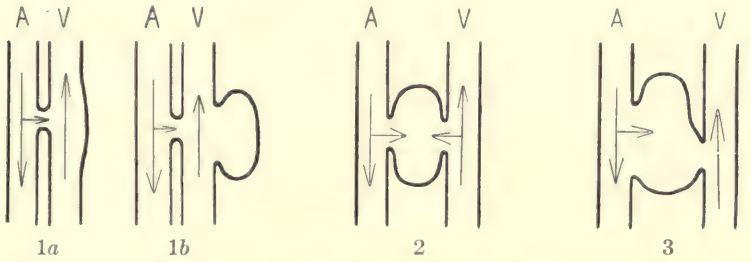


FIG. 249*a*.—THE THREE PRINCIPAL FORMS OF ARTERIOVENOUS ANEURYSM. 1. Arteriovenous fistula (*a*). Arteriovenous aneurysm with venous sac, Varix aneurysmaticus (*b*). 2. Arteriovenous aneurysm with false intermediate sac, Aneurysma varicosum. 3. Arteriovenous aneurysm with arterial sac, secondary arteriovenous aneurysm.

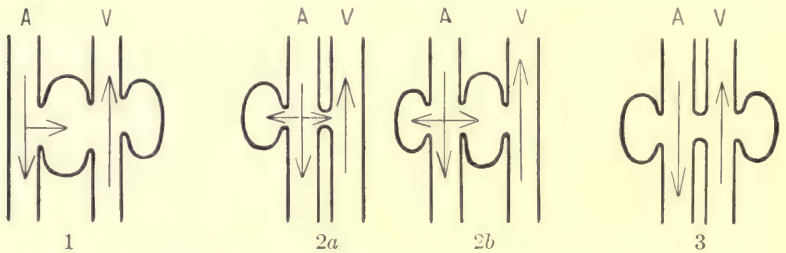


FIG. 249*b*.—SPECIAL FORMS. 1. Arteriovenous aneurysm with false sac and varix on outer side of the vein. Single injury of the artery, double injury of the vein. 2. Arteriovenous aneurysm with direct communication in (*a*) and with a false intermediate sac in (*b*) and with a false arterial aneurysm. (Single injury of vein, double injury of artery.) 3. Arteriovenous fistula, following double injuries of both vessels. The sacs lie opposite each other.

communication is not established directly, as the vein and artery are at first separated by a blood clot. Both the artery and vein communicate with the false, or, according to its position, intermediary sac, which is formed from the blood clot. The wall of the sac consists of the remains of the hæmatoma and thickened connective tissue. It is therefore firmly attached to the surrounding tissues and can be separated with difficulty from nerves, muscles, etc. An aneurysm of this kind may develop at the ends of arteries and veins in an amputation stump.

The **arteriovenous aneurysm** with an **arterial sac** should probably be classified as a varicose aneurysm. It is rare, and develops when a true

aneurysm erodes the wall of a vein and ruptures into it (secondary arteriovenous aneurysm).

Different special forms of arteriovenous aneurysm may develop if, in addition to perforation of corresponding parts of the walls of the artery and vein, one or both vessels perforate at another point. Then the wall of the vein opposite the point of communication with the true or false sac may develop a varix, the artery may develop a false aneurysm, or the varix and aneurysm may be combined in the same case (*vide* Fig. 249).

Most Common Causes of Arteriovenous Aneurysms.—Punctured wounds with sharp instruments (aspirating needle), weapons, or objects (fragment of glass) are the principal causes of arteriovenous aneurysm. [" According to Bramann, of 159 cases of arteriovenous aneurysm, 108 were due to an injury, 56 following phlebotomy, 29 gunshot wounds, 5 contusions which caused no external wound, and 9 were spontaneous. In only four instances was an arteriovenous aneurysm congenital."—Tillmanns' " Text-book of Surgery," Vol. I, p. 534.]

The conditions usually necessary for the production of this form of aneurysm are wounds of the vessel walls lying opposite each other, long and narrow punctured or gunshot wounds with small points of entrance and exit, and repair without inflammation (von Bergmann). In incised and contused wounds rapid agglutination of the edges of the wound is required. If the tunics of the vessel are not completely divided, the tissue at the point of injury must first become necrotic before a communication can be established between the vein and artery. A varicose aneurysm may follow a contusion even when there is no internal wound, as the contused areas in the walls of the vessels agglutinate, and when necrosis occurs an arteriovenous fistula develops. This happened in five of the cases collected by von Bramann.

Order of Frequency in which Vessels are Involved.—According to the statistics of von Bramann and Delbet, the vessels are involved in arteriovenous aneurysms in the following order of frequency: brachial, femoral, popliteal, carotid, arteries of the head, especially the temporal, subclavian, and axillary. This form of aneurysm may also develop spontaneously in the abdominal and thoracic aorta. An arteriovenous aneurysm may develop after injury of the internal carotid artery in the cavernous sinus.

Symptoms.—The symptoms of arteriovenous differ from those of a simple aneurysm, as a part of the arterial blood is poured into the vein. Eddies and whirls in the blood stream, pulsation of the veins, and stasis with subsequent dilatation of the veins are found in this form of aneurysm. Compressibility and expansile pulsation of the tumor are the only symptoms common to the two forms.

The eddies and whirls in the blood stream produce a loud whistling bruit, which indicates that a communication has been established between the artery and vein. This bruit, which is often present immediately after the injury, is most distinct at the point of communication between the vessels, but may be heard for some distance proximalward and distalward to it. It differs from the bruit heard in an arterial aneurysm in that it is continuous and merely increased in intensity during systole, for the arterial pressure is so much greater than the venous that the blood enters the vein continuously, and the rate and force with which it enters is merely increased during systole. It is important to note that this bruit is transmitted along the veins toward the heart. The peripheral transmission is less important, as this is occasionally noted in simple aneurysm. The whirls and eddies also transmit a shock to the vessel wall and the surrounding tissue which is most distinct over the points of communication between vessels, and weaker on either side. The shock imparts to the palpating hand the impression of a trembling motion or a soft thrill (von Bramann, Franz).

The pulsation is not limited to the aneurysmal sac, but extends proximalward and distalward along the vein. Both dilate simultaneously, and the pulsation is more marked in the aneurysmal varix, in which there is no intervening sac, than in the varicose aneurysm, in which there is one. This pulsation is transmitted to the subcutaneous veins only when there is an extensive communication with the deeper veins, as in the arm. If pressure is made upon the artery proximal to the aneurysm the pulsation, bruit, and thrills disappear. If it is possible to press the wall of the sac against a bone so that only the point of communication is closed, the pulse, which was formerly delayed and small, becomes full and strong (von Bramann).

Stasis occurs in the radicles of the diseased veins, as the return flow is interfered with. In the beginning there is merely an œdema and a filling of the subcutaneous veins. Later the skin becomes bluish red in color, the subcutaneous tissue becomes infiltrated and indurated, and elephantiasis develops. The results are nutritional disturbances which are indicated by numbness, itching, a tendency to eczema and ulcer formation, muscular atrophy, and complete loss of function. The nutritional disturbances rarely end in gangrene. As severe nervous symptoms may occur in arteriovenous as in arterial aneurysm, for the sac may exert pressure upon nerves which may also have been wounded at the time the vessels were injured. The severity of these symptoms depends upon the situation of the aneurysm. They are most marked in aneurysms of the neck and legs, least so in aneurysms of the arm and head.

Clinical Course and Results.—An arteriovenous aneurysm should always be regarded as serious (von Bramann). There is no possibility of spontaneous repair, in spite of the fact that the sac may remain unaltered for years. The aneurysm may begin to grow rapidly at any time. The rapid growth may be spontaneous or the result of some external influence. Rupture of the sac, associated with the symptoms already described in discussing arterial aneurysm, may occur at any time. The lesions resulting from the nutritional disturbances may become infected and progressive suppurative inflammation may develop.

Diagnosis.—The diagnosis of arterial and arteriovenous aneurysms, which may be distinguished from each other if the symptoms are closely observed, is not difficult when the symptoms are pronounced, especially if they develop immediately after injury or at the site of a former injury. In differentiating between an aneurysmal varix and a varicose aneurysm, it should always be remembered that the changes in the venous system are always more marked when there is a direct communication between the artery and vein than when there is an intervening sac. Besides, the palpable varix is softer and more easily compressible than is the false sac of the varicose aneurysm. So long as an aneurysm of the thoracic aorta is confined to the thoracic cavity, the diagnosis must be based upon the subjective symptoms and the findings revealed by percussion, auscultation, and the X-ray. When a thoracic aneurysm has eroded the ribs and appears beneath the skin, the latter may become inflamed, and then a diagnosis of an acute or chronic abscess is sometimes made. This same mistake is also made in aneurysms in other parts, when the skin covering them becomes inflamed.

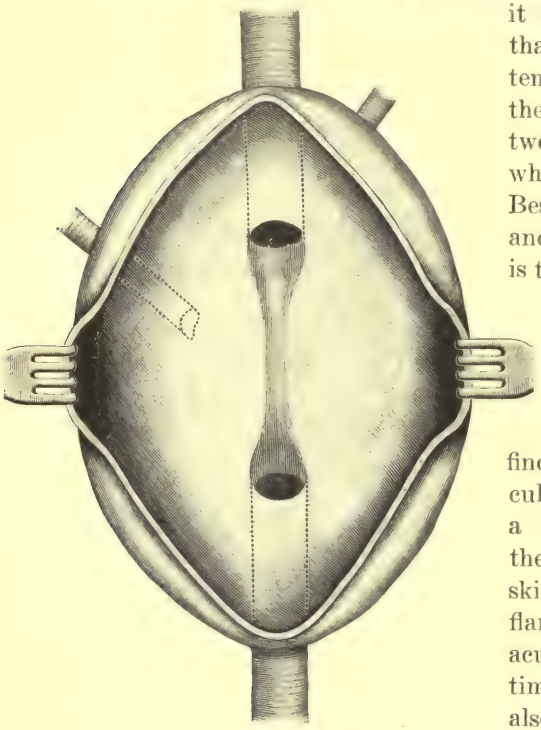


Fig. 250.—The Interior of a Fusiform Aneurysmal Sac, Showing Openings and Groove of Main Vessel and Opening of Collateral Branch. (Matas, Bryant's "Operative Surgery.")

The symptoms of an arterial aneurysm developing spontaneously may be very indefinite, for if small there may be no pressure symptoms, and if the sac is filled with thrombi, no pulsation.

Tumors, infiltrated masses, and abscesses lying over large vessels (e. g., carcinoma of the stomach, horseshoe kidney, inflammatory masses, tuberculous abscesses, and nodes) are to be differentiated from aneurysms by observing carefully the character of the pulsation. These are merely raised or displaced by the pulsation transmitted to them, which is never expansile in character and besides no bruit can be heard.

Cavernous and racemose angiomas are pulsating tumors, and have to be differentiated at times from arteriovenous aneurysms. The vessels of a cavernous angioma are never so full as are those of an arteriovenous aneurysm, and the skin covering such an angioma has a bluish discoloration. The racemose angioma is composed of tortuous, dilated arteries. Sometimes it is very difficult to differentiate between an aneurysm and a pulsating sarcoma.

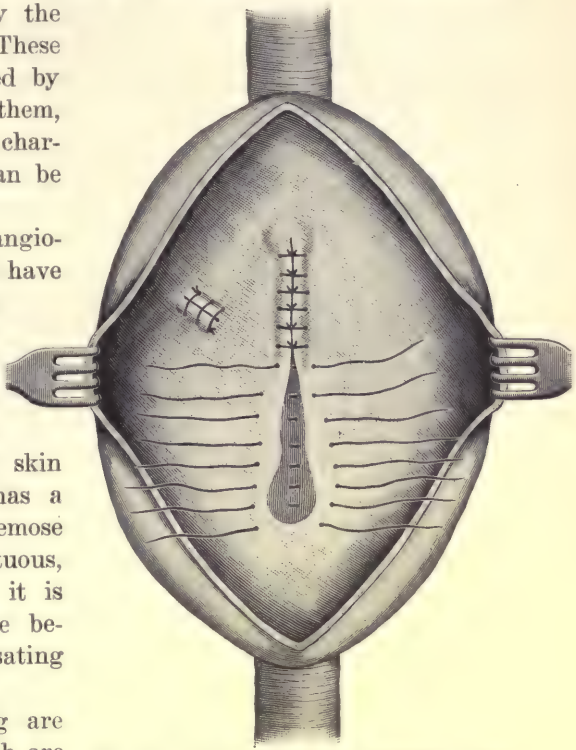


Fig. 251.—The Fusiform Aneurysm. The first row of sutures closing the orifices by fine chromicized catgut or silk. (Matas, Bryant's "Operative Surgery.")

Treatment.—The following are the principal indications which are followed to-day in the surgical treatment of simple and arteriovenous aneurysms: (1) Wherever possible complete or incomplete removal of the sac with double ligation of the artery (vein also in arteriovenous aneurysms) above and below the aneurysm, and ligation of all the communicating lateral branches; (2) restoration of normal conditions, obliterating the aneurysm, without occluding the original lumen of the vessel, by suture (Matas's operation).

Complete extirpation of the aneurysm was first employed in the treatment of arterial aneurysm by Philagrius. After an artificial ischæmia has been produced by the Esmarch method, the upper and lower limits of the sac are exposed, the vessels are doubly ligated and divided, and the sac removed. Frequently, in operating upon large false aneurysms, parts of the sac which are closely adherent to veins and nerves must be left behind. When in such cases it is seen that the sac cannot be enucleated, it should be split open and as much removed as is compatible with

safety. This method has given the best results in the treatment of arteriovenous aneurysms (von Bramann, von Bergmann). The dangers of gangrene following circulatory disturbances induced by this operation (according to Delbet, gangrene occurs in 5.66 per cent of the cases) are to be avoided by applying immobilizing dressings, loose tampons where they seem necessary, and by elevating the extremity. Digital compression applied intermittently for some time before the operation favors the development of a collateral circulation. An attempt should be made to restore the lumen by suturing the cut ends of the vessels, if after removal of a small sac they are not too far removed from each other. In a small saccular aneurysm, aneurysmal varix, and arteriovenous fistula it is possible to restore the lumen of the vessel. After removing the sac or separating the vessels at the point of communication, the defects in the walls should be closed by lateral suture, using the remnants of the sac in making the closure (Matas).

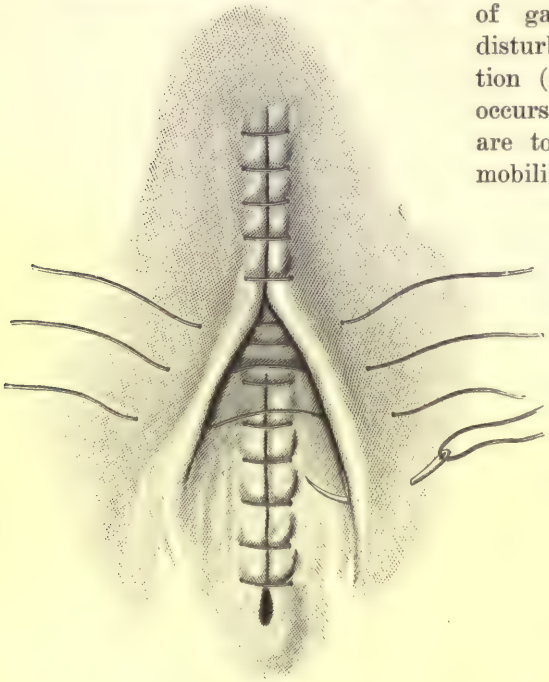


FIG. 252.—THE FUSIFORM ANEURYSM. The second row of sutures. These may be the interrupted or continuous. If floor be rigid the second row may be omitted. (Matas, Bryant's "Operative Surgery.")

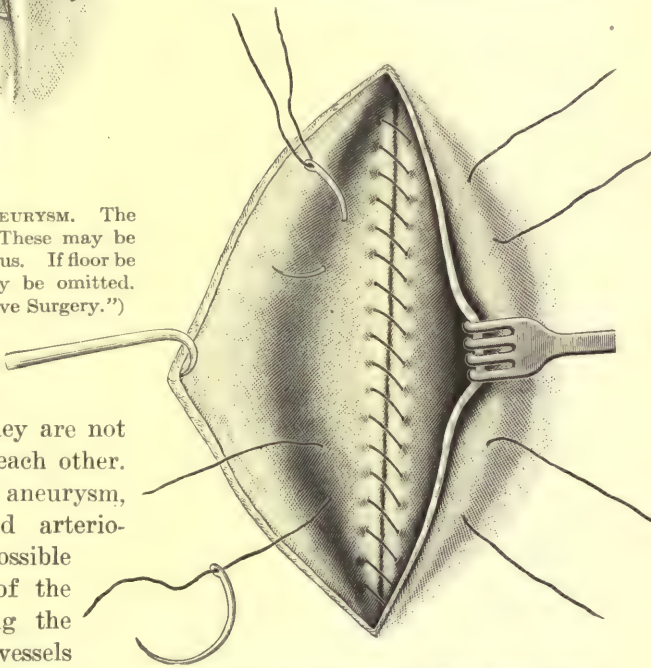


FIG. 253.—THE FUSIFORM ANEURYSM. The second row of sutures (continuous) introduced; the final obliterating sutures passed at either side. On the left, transfixion of floor is made. On the right ends of similar sutures passed through integuments. (Matas, Bryant's "Operative Surgery.")

Incision of the *sac and turning out of the blood clots* after ligation of the artery above and below the aneurysm is a method which dates back to Antyllus. It is used instead of extirpation in the cases in which the latter seems to be impossible because of the size of the aneurysm. After the interior of the sac is exposed, the lateral branches are looked for and ligated and then the cavity of the sac is tamponed.

Proximal ligation of the afferent arterial trunk close to the aneurysm (Anel), or at some distance from it (Hunter), may cause coagulation within and contraction of the sac and result in a cure. Blood may be

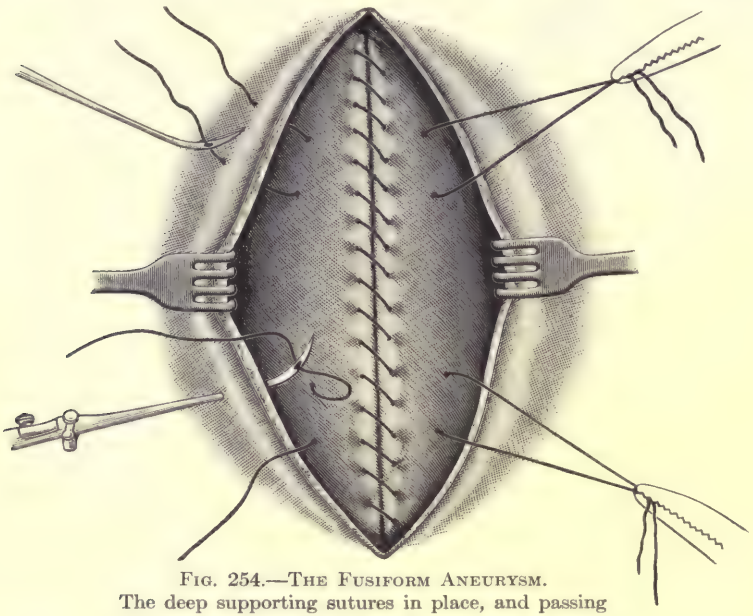


FIG. 254.—THE FUSIFORM ANEURYSM.
The deep supporting sutures in place, and passing their ends through skin and aneurysmal wall.

poured into the sac again when the collateral circulation is established after these operations, and then the aneurysm recurs. There is also a possibility that pieces of thrombi which follow proximal ligation may become loosened and may lodge in the efferent arterial trunk, causing embolic gangrene. Proximal ligation should not be employed in the treatment of an arteriovenous aneurysm, as it is followed quite frequently by gangrene, the arterial blood, when the collateral circulation is established, passing into the vein below the ligature, and enough blood does not reach the peripheral parts to maintain the life of the tissues. According to von Bramann, gangrene developed in six out of thirty-one cases of arteriovenous aneurysms in which proximal ligation of the artery was performed.

Central and *peripheral ligation* of the artery in simple aneurysm (Vigier), of the artery and vein in arteriovenous aneurysms with division of the vessels gives better results than proximal ligation alone.

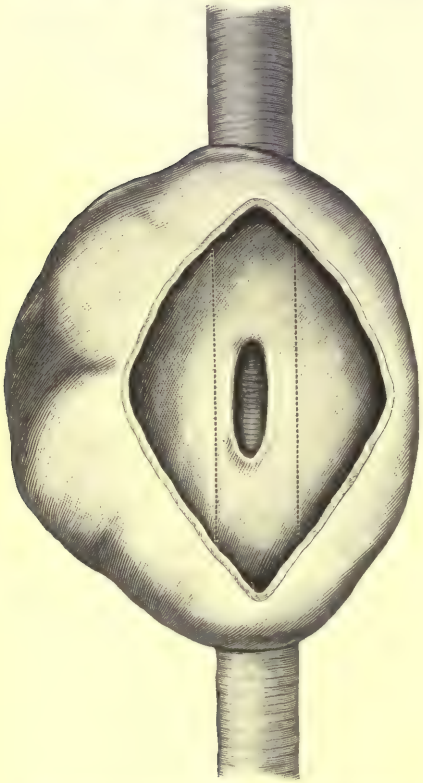


FIG. 255.—THE SACCIFORM ANEURYSM, ITS MAIN ORIFICE AND THE DOTTED OUTLINE OF THE MAIN VESSEL. (Matas, Bryant's "Operative Surgery.")

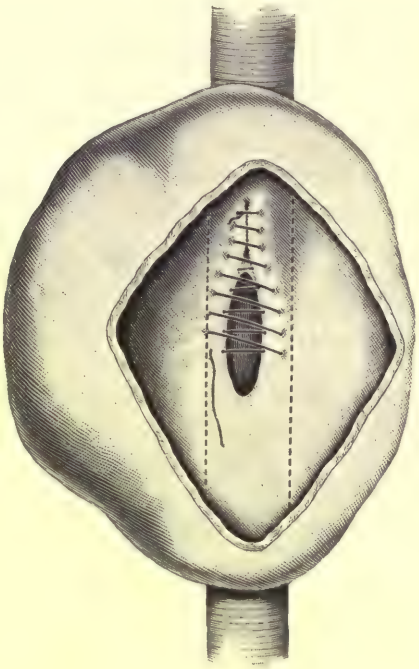


FIG. 256.—THE SACCIFORM ANEURYSM. The closure of main orifice by continuous sutures without special removing of lumen. (Matas, Bryant's "Operative Surgery.")

Even after this operation the aneurysm may recur, as the lateral branches have not been ligated.

Brasdor and Wradrop have recommended peripheral ligation of the artery in the treatment of aneurysms so situated that it is impossible or impracticable to deal with the aneurysm on the cardiac side of the sac. The blood becomes stagnant in the sac after distal ligation, thrombi form which later become organized, resulting in obliteration of the sac and healing. In an aneurysm of the ascending aorta and innominate artery, the right subclavian and common carotid arteries should be ligated. According to H. Jacobs's statistics, thirty-six out of sixty-nine cases treated in this way were cured.

If symptoms (especially bruits) of a pulsating hæmatoma or of an arteriovenous fistula develop after an injury, the vessel or vessels involved should be exposed and closed by lateral arterial or venous suture or ligated depending upon the conditions found. The development of a traumatic or arteriovenous aneurysm may be prevented in this way.

Amputation must be considered in the treatment of large aneurysms of the extremities associated with nutritional disturbances and necrosis, or if embolic gangrene develops.

There are a number of bloodless methods which may be employed in the treatment of inaccessible aneurysms, such as those of the aorta,

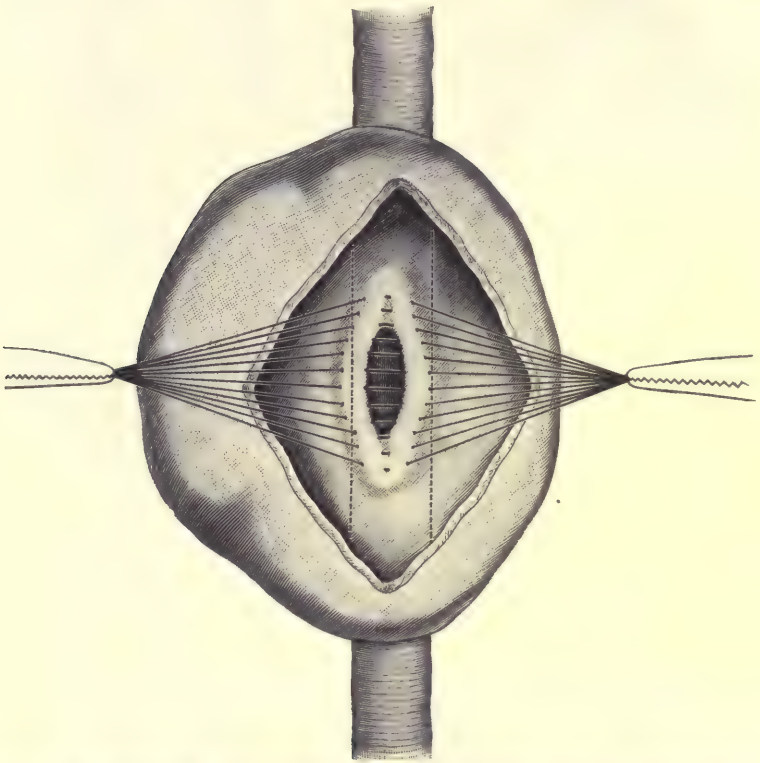


FIG. 257.—THE SACCIFORM ANEURYSM. Closure of the main orifice by interrupted sutures without special removing of lumen. (Matas, Bryant's "Operative Surgery.")

internal carotid, etc., or when operation is contraindicated because of the age or weakened condition of the patient. The object of all these methods is to produce a thrombosis, for when the thrombi become organized the aneurysm becomes smaller and its walls thicker. Of the large number of old and new methods, only two demand consideration.

Compression.—Continuous or intermittent compression of the afferent artery may be employed, some special apparatus or the fingers being used for the purpose. The object of the compression is merely to slow or arrest the arterial stream, not to produce a passive hyperæmia. Cir-

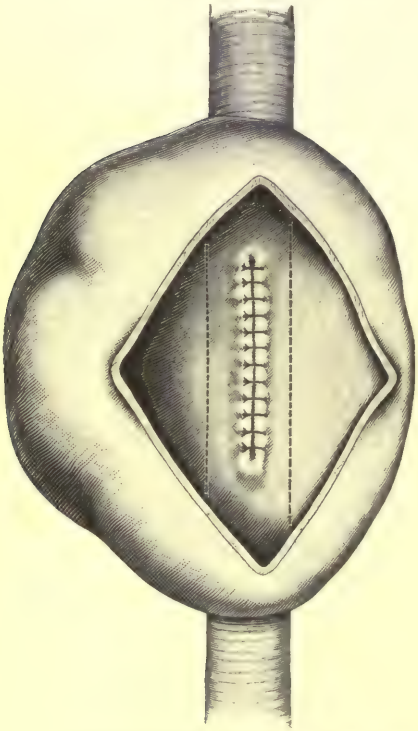


FIG. 258.—THE SACCIFORM ANEURYSM. Obliteration of orifice completed, lumen intact. Operation completed as in fusiform aneurysm. (Matas, Bryant's "Operative Surgery.")

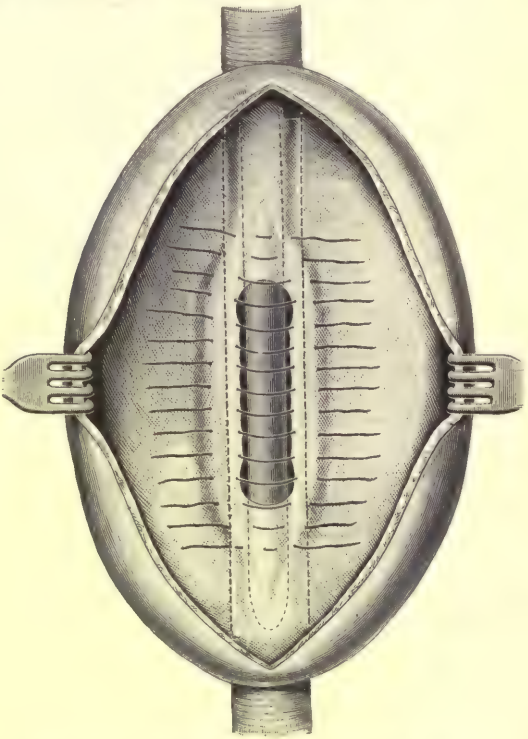


FIG. 259.—THE SACCIFORM ANEURYSM WITH CATHETER INTRODUCED TO MAINTAIN CALIBER OF LUMEN; SUTURES PLACED OVER CATHETER. (Matas, Bryant's "Operative Surgery.")

cular constriction should therefore not be employed for this purpose. Some arterial aneurysms have been observed in which the symptoms disappeared and the sac decreased in size after compression for a number of hours. In other cases intermittent compression has been employed for days and weeks before results were obtained, and then often without success. The object of compression is so to reduce the blood pressure as to permit of coagulation within the sac of the aneurysm. According to Vanzetti, in the treatment of arteriovenous aneurysms by this method the artery should be compressed just above the aneurysm until the pulse disappears, and the vein at the point of communication with the sac at the same time. Compression, combined with rest in bed and im-

mobilization of the extremity, should be employed a short time before operative treatment is instituted. Compression, even if it does not cure the aneurysm, favors the development of a collateral circulation and may prevent subsequent gangrene. In suitable cases forced flexion of the extremity may be employed instead of instrumental or digital compression. The hyperflexed parts are maintained in position by bandages. This method can be employed safely only in the treatment of small aneurysms, as the larger ones are apt to rupture, and in the treatment of aneurysms in certain localities, at the bend of the elbow, in popliteal space, and in the groin.

It should be remembered that there is danger of rupturing large aneurysms when using digital or instrumental compression.

Gelatin Treatment.—Dastre and Floresco demonstrated experimentally that subcutaneous injections of a solution of gelatin increased the coagulability of the blood. Lancereaux and Paulesco (1898) recommended gelatin injections for the treatment of saccular aneurysms. One hundred c.c. of a one or two per cent solution of gelatin (1–2 gm. of white gelatin is dissolved in 100 c.c. of physiological salt solution, and is sterilized for five successive days for one half hour over live steam at 212° F. and heated to 99° F. before being used) should be injected about every week. Beck, among others, cured a large aneurysm of the aorta in two months by this treatment. This method, which promised much, has been disappointing. If the heart is weak the increased coagulability of the blood may lead to the formation of thrombi in the large veins (iliac femoral), and if the sterilization is not thorough and reliable, tetanus may follow the injection (*vide* Bass).

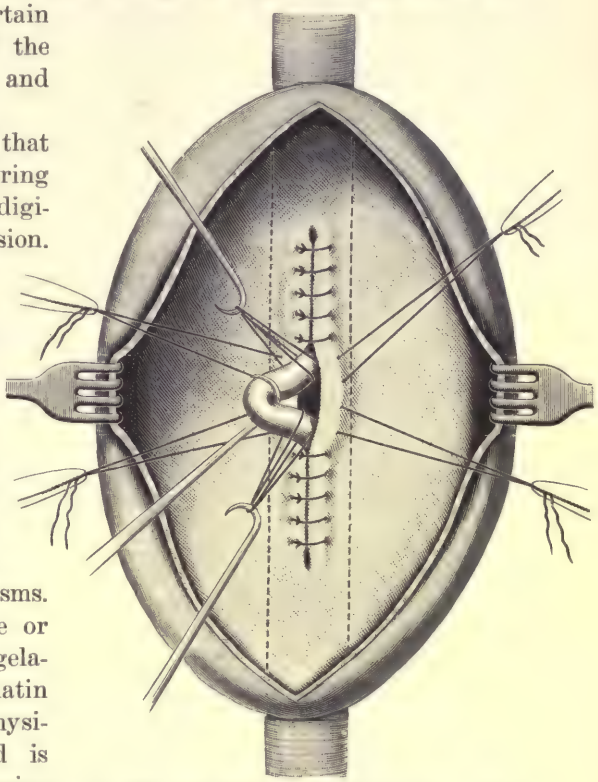


FIG. 260.—THE SACCIFORM ANEURYSM. The removal of catheter before final closure of the main channel. (Matas, Bryant's "Operative Surgery.")

Injections of chemical agents into the sac, and the application of

elastic bandages about the extremity, are frequently followed by gangrene and should not be employed. Acupuncture, according to Velpeau (insertion of hot needles into the sac), and galvano-puncture (introducing gold or steel wire through a needle or canula into the sac and passing the galvanic current through them) are not very often indicated, as the treatment is often not successful and is associated with the dangers of hæmorrhage, inflammation, and separation of thrombi.

If the aneurysm is inoperable, the patient should be kept in bed or the extremity should be immobilized. Sudden increase in blood pressure, such as follows physical effort, excitement, and the use of alcoholic drinks, should be avoided. Treatment by starvation and blood-letting, as prescribed in Valsalva's method, should be employed. Digitalis and tincture of strophanthus may be used to quiet the heart. Iodid of potassium and sodium have a favorable influence upon the local and general conditions.

LITERATURE.—*Bass.* Erfolge und Gefahren der Gelatineapplikation. Zentralbl. f. d. Grenzgebiete, 1904, p. 118.—*v. Bergmann.* Zur Kasuistik des arteriell-venösen traumatischen Aneurysma. Arch. f. klin. Chir., Bd. 69, 1903, p. 515.—*v. Bramann.* Das arteriell-venöse Aneurysma. Arch. f. klin. Chir., Bd. 73, 1886, p. 1.—*Delbet.* Maladies chir. des artères. Traité de chir. le Dentu et Delbet. Paris, 1897.—*Eppinger.* Pathogenesis, Histogenesis und Aetiologie der Aneurysmen. Arch. f. klin. Chir., Bd. 35, 1887, Suppl., p. 1.—*Fr. Fischer.* Krankheiten der Lymphgefässe, Lymphdrüsen und Blutgefässe. Deutsche Chir., 1901.—*Franz.* Klinische und experimentelle Beiträge betreffend das Aneurysma arteriovenosum. Arch. f. klin. Chir., Bd. 75, 1905, p. 572.—*v. Frisch.* Beitrag z. Behandl. periph. Aneurysmen. Arch. f. klin. Chir., Bd. 79, 1906, p. 515.—*Jacobsthal.* Beitr. zur Statistik der operativ behandelten Aneurysmen. I. Das Aneurysma der Art. anonyma. Deutsche Zeitschr. f. Chir., Bd. 63, 1902, p. 550. II. Das Aneurysma der Art. subclavia. Ibid., Bd. 68, 1903, p. 239.—*Malkoff.* Ueber die Bedeutung der traumatischen Verletzung von Arterien (Quetschung, Dehnung) für die Entwicklung der wahren Aneurysmen und der Arteriosklerose. Zieglers Beiträge zur path. Anat., Bd. 25, 1901, p. 431.—*Matas.* An Operation for the Radical Cure of Aneurism. Transact. of the Americ. Surgical Assn., vol. 20, 1902.—*Orth.* Lehrbuch der speziellen pathol. Anatomie, I.—*v. Schrötter.* Die Erkrankungen der Gefässe. Nothnagels Handbuch d. spez. Path. u. Ther., Bd. 15, Part III, Wien, 1901.—*Sorgo.* Behandlung der Aneurysmen mit subkutanen Gelatineinjektionen. Zentralbl. f. Grenzgeb., 1899, p. 10.—*Thoma.* Untersuchungen über Aneurysmen. Virchows Archiv., 111-113, 1888;—Elastizität gesunder und kranker Arterien. Ibid., Bd. 116, 1889, p. 1.

(c) PHLEBECTASES, VARICES

By phlebectases or varices is understood a permanent dilatation of the walls of large and small veins. The former term is used more frequently to designate the fusiform, cylindrical, tortuous (cirsoid) dilatations, while the latter is applied to the circumscribed bulgings of the vein wall (varices), which still maintain their connection with the vein by a broad or narrow neck. There are a number of transitional forms, so

that both terms are usually employed with about the same general significance.

Causes of Varicose Veins.—There are a number of etiological factors which contribute to the development of varicose veins. Besides the mechanical factors which interfere with the venous circulation and therefore increase the pressure within the veins, the lessened resistance of the walls of the veins and insufficiency of the valves, which may be congenital or secondary to inflammatory changes, must be considered. Usually a number of different etiological factors are combined, one following and accentuating another; for example, if the valves of the veins of the lower extremities have undergone contraction and become insufficient, the weight of the entire column of blood from the inferior vena cava down is thrown upon the wall of the vein. If the reverse happens and the walls of the vein become distended by the weight of the column of blood, the valves become insufficient and are no longer able to break the column of blood and assist in venous circulation. If the stasis is long-continued, the walls of the veins become inelastic and yielding, the circulation in the vasa vasorum is interfered with and the nutrition of the tissues of the vein wall gradually is impaired, leading to a dilatation of the vein with relative insufficiency of the valves and venous stasis. According to Ledderhose, too much importance has been attributed to the valves in assisting in and maintaining venous circulation. Most authorities, however, at the present time attribute to the valves a very important function.

FIG. 261.—RESECTED PIECE OF THE LONG SAPHENOUS VEIN (FILLED WITH PARAFFINE). 1, cylindrical; 2, fusiform; 3, tortuous phlebectases; 4, varix.

Pathological Changes in Varicose Veins.—Some parts of a varicose vein may be practically normal, while in other parts the muscular and elastic elements disappear, being replaced by a fibro-cicatricial tissue, while in still other parts (especially where the pouchlike dilatations occur) the vein wall is thin and atrophic. The veins are more or less adherent to surrounding structures, the adhesions being partly due to nutritional disturbances, partly to inflammatory changes in the peri-

vascular tissues. The slowing of the blood stream and the proliferation of the intima predispose to the development of thrombi. If the thrombi become calcified, *vein stones* or *phleboliths* are formed. A varix, which may become as large as a hen's egg, may become constricted and separated from the vein wall at the point at which it formerly communicated. In this way a blood cyst may develop. Large, tortuous, and convoluted veins may be in direct communication with one another, as the walls of the vein when in contact may undergo a pressure necrosis and a communication may be established (anastomosing varices).

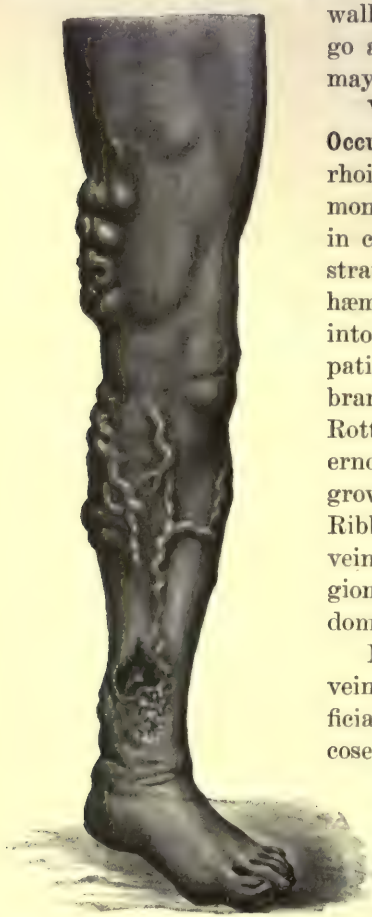


Fig. 262.—Varicose Veins of the Lower Extremity.

Veins Most Commonly Involved—Age, Sex, Occupation.—Hæmorrhoids (dilated hæmorrhoidal veins or plexuses) are the most common type of varicose veins. Normally, even in children, small dilatations may be demonstrated in the veins about the anus (annulus hæmorrhoidalis), which become transformed into varices as the result of chronic constipation and inflammation of the mucous membrane. Some authors (Reinbach, Gunckel, Rotter, Ziegler) regard hæmorrhoids as cavernous angiomas and classify them as new growths, while others (Borst, Kaufmann, Ribbert) regard them merely as varicose veins. [Hæmorrhoids are histologically angiomas in which the venous elements predominate.]

Next in order of frequency are varicose veins of the lower extremity, both the superficial and deep veins being involved. Varicose veins are most common in individuals of middle age, whose occupation requires them to stand a great deal, in women who have borne children, in patients with pelvic tumors, and in young people with a congenital weakness of the vein walls. Blue nodules, sacculated and tortuous dilatations, and convolutions are seen in the course of the long and short saphenous veins, over which the skin is more or less thinned. These empty when pressure is made upon them or the extremity is elevated, leaving deep grooves in the thinned skin. When the upright position is assumed again and the pressure is released, the blood flows back into the veins

from above, and the veins do not fill from below as the valves are insufficient. [This test was first used by Trendelenburg and may be employed in the following way: The patient lies down and the veins are allowed to empty. Pressure is then made upon the saphenous vein at the saphenous opening, and maintained while the patient is assuming the upright position. When the pressure is released the column of blood drops back from above, showing that the valves are insufficient.]

The small veins of the skin are red and injected. Varicose veins in the upper extremity are rare, and are usually due to pressure of tumors upon the axillary or subclavian vein, or are associated with arteriovenous or cirsoid aneurysms.

A varicose condition of the pampiniform plexus is known as a varicocele. Varicoceles are common in young adults. Varicose veins also develop in later adult life within the broad ligament, the prostate, about the neck of the bladder and the external genitalia, in the utero-vaginal, vesical, and pudendal plexuses. The veins of the abdominal wall surrounding the umbilicus dilate to form the caput Medusæ when the portal circulation is obstructed by thrombosis or in cirrhosis of the liver, aiding in the establishment of a collateral circulation. The subcutaneous veins of the thorax become dilated in tumors of the mediastinum.

Results of Varicose Veins.—The results of varicose veins are *circulatory* and *nutritional disturbances.* These are most pronounced when the veins of the lower extremities are involved. The skin becomes thinned and atrophic, susceptible to all kinds of infection, injuries, and necrosis (*vide* p. 506). Varicose ulcers, eczema, and thrombophlebitis are frequent. An œdema, varying in degree, follows the passive hyperæmia and leads to a gradual thickening and induration of the skin and subcutaneous connective tissues (elephantiasis phlebectatica). Weakness of the legs and fatigue are common, and are due in part to the circulatory disturbances, in part to the fibrous myositis with secondary degeneration of the muscle fibers. Cramplike muscular contractions and neuralgic pains are of frequent occurrence when the deeper veins are involved. Submucous varices may cause an atrophy of the mucous membrane, a varicocele, atrophy of the testicle.

Dangers of Varicose Veins.—The dangers of varicose veins are *hæmorrhage* and *thrombophlebitis.* Hæmorrhage follows rupture of a pouchlike dilatation of the vein wall and the atrophic skin or mucous membrane covering it. It is usually due to a sudden increase in venous pressure resulting from the imperfect application of an Esmarch constrictor or the dependent position of the extremity. Such a hæmorrhage may prove fatal, as a column of blood extending to the right auricle is opened near its lower end.

Hæmorrhoids rupture and bleed very frequently (therefore the name). Occasionally varicose veins rupture into the hollow viscera (rupture of œsophageal varices in cirrhosis of the liver and of varices of the brain) terminating fatally. A sudden, frequently painful, swelling develops when varicose veins in a muscle rupture.

Diagnosis.—The diagnosis of superficial varicose veins is difficult only when the dilatations are limited to a small part of the vein and are circumscribed. In these cases a diagnosis of a cavernous hæmangioma may be made. Deep varicose veins give rise to no definite symptoms. The dilated, pulsating veins occurring in arteriovenous aneurysm offer no difficulty in diagnosis if a careful examination is made. The varices occurring over the saphenous opening and varicocele are sometimes mistaken for femoral and inguinal hernia respectively, but differ from hernia in that the swelling disappears so readily when pressure is made or when the patient lies down, and recurs so readily when the patient stands up.

Treatment.—The treatment which should be instituted depends upon the cause, the situation, and the complications of the varicose veins.

Varicose veins frequently subside after tumors (which have exerted pressure upon the principal vein and have caused an increased venous pressure) have been removed. Ligation of the long saphenous vein at the saphenous opening, as suggested by Trendelenburg, breaks the long column of venous blood and prevents pressure upon the wall of the vein.

Elastic bandages properly applied and elevation of the extremities have a favorable influence upon varicose veins of the extremities. A suspensory improves the circulation in a varicocele, and the pain and discomfort usually soon disappear after it is used.

Large varicose veins and varicoceles should be extirpated after the veins have been exposed and ligated. In varicose veins of the leg extensive and thorough resection of the diseased veins (Madelung) or ligation and resection of the long saphenous vein at the saphenous opening are recommended (Trendelenburg). [C. H. Mayo has devised a very ingenious instrument, called a "vein stripper," which permits of a subcutaneous removal of the greater part of the varicose vein. It is a long instrument, provided with a steel eye. The vein is exposed above through a small transverse incision, cut and ligated, and then threaded upon the instrument. The vein is then separated from the tissues, and its collaterals are broken by gently forcing the "stripper" along the vein. Another small incision is then made over the end of the instrument, the distal portion of the vein is ligated, and the part which has been separated is removed.] Hæmorrhoids should be removed with the actual cautery or transfixed at the base with heavy silk and ligated.

Bleeding from varicose veins subsides if the extremity is elevated

or mild compression is exerted by a bandage. Hæmorrhage from hæmorrhoids generally ceases when the prolapsed, strangulated masses are reduced. Extirpation of the larger ruptured varices is advised. The treatment of other complications, such as thrombophlebitis and varicose ulcers, will be found in the chapters devoted to these subjects.

LITERATURE.—*Fr. Fischer.* Krankheiten der Lymphgefässe, Lymphdrüsen und Blutgefässe. Deutsche Chir., 1901.—*F. Fraenkel.* Ueber die Behandlung der Varizen der Unteren Extremität durch Ausschälung nach Madelung. Beitr. z. klin. Chir., Bd. 36, 1902, p. 547.—*Kashimura.* Die Entstehung der Varizen der Vena saphena in ihrer Abhängigkeit vom Gefässnervensystem. Virchows Arch., Bd. 179, 1905, p. 373.—*Ledderhose.* Die Bedeutung der Venenklappen und ihre Beziehungen zu den Varizen. Deutsch. med. Wochenschr., 1904, p. 1563.—*v. Schrötter.* Erkrankungen der Gefässe. Nothnagels Spez. Path. u. Ther., Wien, 1901.—*Schwarz.* Maladies chir. des veines. Traité de chir. le Dentu et Delbet. T. IV, p. 349. Paris, 1897.

(d) THROMBOSIS AND EMBOLISM

THROMBOSIS

The coagulation of blood within the vessels during life is known as thrombosis, and the resulting solid mass as a thrombus (from the Greek θρόμβος, meaning coagulum).

Varieties of Thrombi and Histological Changes Occurring in Thrombosis.—Red, white, and mixed thrombi have been distinguished since Zahn first described the microscopic changes occurring in thrombosis of the mesenteric vessels of a frog. The color of a thrombus depends upon the number of red blood corpuscles it contains. The red thrombus contains, besides granules and threads of fibrin, all the component parts of the blood, and is formed when stagnant or slowly flowing blood coagulates. Thrombi formed from blood in circulation, which not infrequently occur upon the internal surface of the heart and blood vessels, are composed mostly of fibrin, with a variable number of the colorless elements of the blood, and contain sometimes a few red cells. White and mixed thrombi are formed when the circulating blood coagulates. [The following description of the formation of thrombi is found in Ziegler's "General Pathology," pp. 117 and 118: "The formation of thrombi in circulating blood may be observed distinctly under the microscope, in suitable subjects, both in warm-blooded and cold-blooded animals, and in this line it is more particularly the observations of Bizzozero, Eberth, Schimmelbusch, and Löwit which have led to very weighty conclusions. When the blood flows through a vessel with its normal velocity, you may see under the microscope (Bizzozero, Eberth, and Schimmelbusch) a broad, homogeneous, red stream in the axis of the blood vessel, while at the sides lies a clear zone of blood-plasma free from red blood corpuscles.

" This may be observed in both arteries and veins and in the larger capillaries, but is best seen in the veins. In the capillaries just large enough to permit of the passage of the blood corpuscles, this differentiation into an axial and peripheral stream does not hold. In the axial stream the different constituents of the blood are not recognizable; in the peripheral stream, however, isolated white blood corpuscles appear from time to time, and these may be seen to roll slowly along the vessel wall. If the blood current becomes retarded to about the degree which allows the observer to make out indistinctly the blood corpuscles of the axial stream, the number of white blood corpuscles floating slowly along in the peripheral zone, and adhering also at times to the vessel wall, becomes increased, and they finally come to occupy this zone in considerable numbers. If the current be still further retarded so that the red blood corpuscles become clearly recognizable, then in the peripheral zone alongside of the white blood corpuscles appear blood platelets, which increase more and more in number with the progressive retardation of the flow, while the number of leucocytes becomes again diminished. When total arrest of the blood current finally occurs, a distinct separation of the corpuscular elements in the lumen of the vessel follows.

" When, in a vessel in which circulation is retarded, the intima is injured at a certain point by compression or by violence, or by chemical agents, such as corrosive sublimate, nitrate of silver, or strong salt solutions, and the lesion of the vessel wall is of such a character that it does not cause arrest of the blood current, we may observe (Bizzozero, Eberth, Schimmelbusch) blood plates adhering to the vessel wall at the injured point, and before long they cover the site of the injury in several layers.

" Frequently more or less numerous leucocytes or colorless blood corpuscles become lodged in the mass (Bizzozero), and their number is proportionate to their abundance in the peripheral zone. Under some circumstances the number of leucocytes may be very considerable, and they may largely cover over the accumulation of blood plates. In case of great irregularity of the circulation or of extensive lesions of the vascular wall, red blood corpuscles also may separate from the circulation and become adherent to the intima or to a layer of leucocytes previously deposited upon it. Not infrequently portions of the separated mass are swept away, in which case a new deposit of blood plates is formed. Through a long-continued deposition of the elements of the blood the vessel may finally become completely closed.

" Should a blood vessel suffer a lesion, as above described, while the current of blood within it still remains swift, there is no adherence of blood plates or of blood corpuscles. When at any point blood plates have become adherent in considerable numbers, after a time they be-

come coarsely granular at the center, granular or homogeneous at the periphery, and finally become fused into one compact mass. The final result of the process is the formation of a colorless blood-plate thrombus, within which more or less numerous white blood corpuscles may be imprisoned. Eberth designates the sticking together of the blood plates by the term conglutination; their final fusion into a coherent thrombus he calls viscous metamorphosis.

"If we compare the observations of Bizzozero, Eberth, and Schimmelbusch, as well as the recent observations of Löwit, on warm-blooded animals with the histological findings in thrombi from the human subject, we are warranted in drawing the conclusion that the formation of thrombi in the circulating blood of man proceeds in a way similar to that observed in the lower animals, and we judge that their formation is directly dependent upon two causes: (1) Upon a retardation of the blood current or other disturbance of the circulation, such as the formation of eddies which would direct the blood plates against the vascular wall, and (2) upon local changes in the wall of the vessel. Probably, too, thrombosis is favored by pathological changes in the blood. From the variety of conditions under which thrombosis occurs in man we must assume either that now one and again another of these causes plays the principal part in the formation of thrombi, or that all these may concur equally in the process; and, on the other hand, that one of the causes alone is not ordinarily sufficient to cause thrombosis."]

Origin of Blood Plates, Fibrin Ferment, etc.—The finer processes of thrombus formation are not well understood. The origin and significance of the blood plates discovered by Bizzozero are not clear. According to the prevailing view they are formed from degenerating red blood corpuscles, yet it is possible that they have no single source (Grawitz). Liberation of fibrin ferment or thrombin (Alex. Schmidt) precedes coagulation. It apparently is derived from degenerating cells (white and red blood corpuscles and endothelial cells) and acts upon fibrinogen, an albuminous substance in the blood plasma. According to Arthus and Pagès a third factor, a calcium salt, must be present before coagulation can take place. Pekelharing believes that calcium is transferred by the fibrin ferment to the fibrinogen, and that the latter, which was previously soluble, undergoes a chemical metamorphosis resulting in the formation of an insoluble calcium-albumin compound, fibrin.

Factors Concerned in Thrombus Formation.—Three different factors are concerned in thrombus formation: (1) Slowing of the blood current, (2) changes in the vessel wall, and (3) alterations in the composition of the blood.

Slowing of the blood stream, following the general circulatory disturbances due to cardiac asthenia which occur in a number of different

diseases, is the main factor in the development of the so-called *marantic* (marasmic) *thrombi.* Local causes interfering with circulation are narrowing of the lumen of the vessel caused by disease of the vessel wall (arteriosclerosis), or compression of the vessel by tumors, displaced fragments of bone, dislocated bones and constricting bandages, and the development of whirls and eddies in aneurysms and in pouchlike dilatations in the walls of varicose veins.

A very insignificant injury of the endothelium lining a vessel may be followed by thrombus formation, and of course a thrombus is much more apt to form when the injury to the vessel wall is more extensive. Large arteries are closed spontaneously when crushed or lacerated by the separation and rolling in of the intima. [The rolling up of the intima prevents hæmorrhage after crushed and lacerated wounds of even the largest arteries. We have seen an axillary artery completely plugged by endothelium in a crushing injury of the shoulder. At least an inch of the intima could be unrolled when the artery was divided after being ligated. The endothelium had formed a complete plug for the vessel.] Spontaneous healing of small wounds of vessels begins with thrombus formation (*vide* Injuries of Vessels). Changes in the vessel walls due to chemical and thermal agents, to diseases, such as arteriosclerosis, suppurative and tuberculous inflammation favor thrombus formation. Thrombus formation may also follow penetrating wounds (e. g., needles) and infiltration of the vessel wall by tumor masses.

Alterations in the *composition* of the *blood* may increase its coagulability. The increased coagulability of the blood in general infections (general pyogenic infections, typhoid fever, influenza, etc.), in diseases of the blood (chlorosis), and after extensive burns is probably due to the increase in fibrin ferment resulting from an increased destruction of cells.

Mural and Obturating Thrombi.—Parietal, or mural, and obturating thrombi are described, depending upon their relation to the vessel containing them. Fibrin may be deposited upon a parietal thrombus, which then enlarges until it may become an obturating thrombus. A thrombus either remains limited to the point at which it develops, in which case it is firmly attached, or gradually grows as new masses of fibrin are deposited, and extends from a small into a larger vessel or vice versa; for example, a thrombus developing in a small vein of the foot may extend to the inferior vena cava. Such a thrombus, however, is never firmly attached throughout to the intima. Upon section thrombi often have a streaked appearance and irregular markings, brighter and darker areas alternating. Not infrequently it happens that a red thrombus is superadded to a white or mixed thrombus as the coagulation began in circulating blood, and after the vessel is occluded the blood

becomes stagnant and the whole mass then coagulates. Thrombi forming in aneurysmal sacs are frequently laminated, for thrombosis is not continuous and progressive, but occurs at intervals, so that the newly formed layers of coagulum do not become firmly attached to the old.

Changes Occurring in Thrombi.—Thrombi may undergo a number of different changes, such as *contraction, calcification, simple* and *septic softening,* and *organization.* A thrombus in the beginning is soft and contains fluid, but after a time the fibrin contracts, expressing the fluid, the cells enclosed within the meshes of the fibrin degenerate, and the mass becomes dry and firm. A vessel which has been completely closed may become patent again when the thrombus contracts. If lime salts are deposited in the thrombus or the mass which replaces it, vein or artery stones (phleboliths and arterioliths) are formed. When a thrombus undergoes simple softening the central portion becomes transformed into a grayish red, caseous, degenerating mass which, after the external layers degenerate, is broken up and discharged into the blood stream, giving rise to emboli. Suppurative or putrefactive softening is due to inflammation of the vessel wall, resulting from the invasion of pyogenic or putrefactive bacteria. It may be followed by the discharge into the blood stream of numerous infected emboli. The most favorable change in a thrombus is organization. A vascular, germinal tissue which develops from proliferating endothelium invades and replaces the thrombus which becomes transformed into firm connective tissue, and the vessel is either permanently closed or its wall is thickened.

Symptoms of Thrombosis.—The symptoms of thrombosis are not pronounced unless one of the larger arteries is completely occluded. Nutritional disturbances then develop which may end in gangrene unless a sufficient collateral circulation is established. Stasis is the principal symptom of venous thrombosis. Other symptoms due to separation of particles of the thrombus and subsequent embolism are frequent.

Thrombi develop in arteries after injuries of the vessel wall, in aneurysms, in acute inflammation or chronic diseases of the vessels, and after the lodgment of emboli originating from thrombi within the heart or larger vessels.

Venous thrombi develop very frequently in phlebitis and in chronic diseases in which cardiac weakness and the absorption of toxins cooperate in producing conditions favoring thrombosis. The veins of the lower extremity are involved most frequently, for the circulation is not only poor in these veins if they are dilated, but phlebitis is also common.

When venous thrombosis occurs there may be found along the course of the subcutaneous veins hard, painful, tortuous cords and considerable œdema. In thrombosis of the femoral vein there develops, besides the hard cord the size of a thumb, cyanosis and marked œdema which fre-

quently becomes chronic, resulting in permanent enlargement of the limb. Thrombi may develop in the veins of the pampiniform plexus of the female after infections and operations, and extend by way of the internal iliac into the femoral vein. Œdema of one or both legs then develops, and the resulting clinical picture resembles that known as phlegmasia alba dolens, following puerperal infections. Suppurative otitis media is frequently the cause of thrombosis of the sigmoid sinus. The thrombus forming in this sinus may extend to communicating sinuses and to the internal jugular vein. Thrombosis of the veins of the mesentery may follow internal strangulation or the incarceration of intestinal loops or omentum in a hernia. The thrombi may then extend to the portal vein, causing marked stasis in the territory drained by the radicles of this vein. Large veins, such as the superior and inferior vena cava and subclavian, may be closed by thrombi extending into them from smaller radicles or by thrombi caused by the pressure of large tumors or aneurysms. If a sufficient collateral venous circulation is established the œdema gradually subsides. Gangrene develops only when all the veins draining an area or organ become closed by thrombi.

EMBOLISM

The dangers of embolism are associated with thrombosis. Particles of thrombi may be broken off by trauma, separated by violent movements, or discharged spontaneously when the thrombus undergoes simple or puriform softening. The advancing end of a thrombus which has extended from a small vein or artery into the lumen of a larger vein or artery may be separated and carried away into the blood stream.

Lodgment of an Embolus.—An embolus originating in the left heart or one of the larger arteries may be carried in the blood stream until it either lodges at the point of bifurcation of an artery, where it may remain attached as a saddle-shaped embolus occluding both branches, or may be carried along until the lumen of the artery is so reduced that it becomes caught. If an important artery is occluded, gangrene of the part supplied by the artery accompanied by violent symptoms frequently follows, as a collateral circulation sufficient to provide for the nutrition of the tissues is not established rapidly enough (*vide* p. 497). Infected emboli may cause arteritis and embolomycotic aneurysms.

Pulmonary Embolism.—Venous emboli, originating not only in large thrombosed vessels, but also in small veins adjacent to insignificant injuries and inflammatory foci (e. g., fracture of the fibula, furuncle), are much more frequent and are usually more dangerous than are arterial emboli. They pass from the vessel into the right heart, thence into the pulmonary arteries, occluding the principal artery or its branches. If the principal artery or one of its large branches is occluded, marked

dyspnœa develops suddenly, the heart becomes rapid, weak, and soon exhausted, and death occurs. If one of the less important branches becomes occluded—this occurs especially in the right lower lobe—the symptoms of hæmorrhagic infarct soon develop.

In rare cases an embolus may pass through a patent foramen ovale into the general circulation (paradoxical embolus) or an embolus in a large vein may be carried backward (retrograde embolism) in a direction opposite to the current, when there is venous stasis and the pulse wave is transmitted to the blood in the veins (Ribbert).

Metastatic lung abscesses may be caused by infected emboli. Emboli arising from inflammatory foci in the lungs (especially tuberculous foci) may pass into the general circulation and frequently produce infarct-shaped foci in the viscera.

Diagnosis.—The diagnosis of thrombosis can be made only when the large vessels are involved. Chronic œdema resulting from venous thrombosis may be easily mistaken for œdema due to other causes.

Treatment.—Rest in bed and immobilization with elevation of the affected part should be maintained for many weeks—for at least three. These measures favor the contraction or organization of the thrombus and the establishment of a collateral circulation. If the œdema persists, a bandage or elastic stocking exerting mild compression should be worn. Absolute rest is the best protection against embolism, especially against pulmonary embolism, which is always of the gravest significance. Large doses of morphin are often indispensable in quieting the patient. Cardiac stimulants should not be given unless cardiac weakness becomes serious, as the increased force of the heart beat may easily separate and set free particles of a thrombus.

Sudden death from pulmonary embolism is a constant menace, even when convalescence from inflammatory processes adjacent to the veins of the abdomen and pelvis (especially in appendicitis and inflammation of the adnexa and after operations performed for the relief of the same) is well advanced. Improvement of the general condition and of the heart and complete rest in the recumbent position are the only methods by which this serious accident can be prevented.

LITERATURE.—*Grawitz.* Klinische Pathologie des Blutes. Berlin, 1902. Die Blutplättchen, p. 128.—*v. Schrötter.* Erkrankungen der Gefässe. Wien, 1901.—*Ziegler.* Thrombose. Eulenburgs Realenzyklopädie, 3. Aufl.

(e) LYMPHANGIECTASES

Lymphangiectases of the thoracic duct and the larger lymphatic vessels occur especially as the result of the pressure of tumors, and are of importance only in those cases in which they rupture into the pleural or peritoneal cavities, causing chylothorax and chylous ascites.

Causes of Lymphatic Varices—Lymph-Œdema.—Varices develop in the lymphatic vessels of the skin and subcutaneous tissue after frequently recurring or continued inflammations followed by thrombosis and obliteration of the vessels (in habitual erysipelas, recurring lymphangitis, and invasion of the lymphatic vessels by the filaria sanguinis); occasionally after extirpation of suppurating inguinal lymph nodes; after extensive crushing injuries and phlegmons. Not infrequently dilatation of lymphatic vessels resulting from inflammation is associated with pachydermia. If the cutaneous lymphatics are involved, the skin becomes swollen, the boundaries of the swelling being indistinct, and covered with small, closely set vesicles which never become larger than a pea. The skin is also filled with dilated, tortuous lymphatic vessels. If pressure is made upon the swollen area, the fluid, as in œdema, may be forced into the surrounding tissues, and a pit which slowly disappears remains when the pressure is removed.

If the larger vessels in the subcutaneous tissues are dilated, tortuous cords resembling angleworms may be seen. The skin over these is covered with small vesicles and presents the signs common to a lymph-œdema. [The characteristic appearance of lymph-œdema is seen in the pigskinlike changes associated with carcinoma of the breast. The peculiar appearance of the skin in these cases is due to a lymph-œdema following occlusion of the lymphatic vessels by carcinoma cells.]

Clinical Course and Diagnosis.—The growth of a lymphangiectatic swelling is very slow or, after acute inflammatory processes, intermittent. It is scarcely possible to differentiate less extensive lymphangiectases from a lymphangioma. All congenital dilatations of lymphatic vessels should be classified with lymphangiomas. They can scarcely be mistaken for varicose veins, as the bluish color of the skin indicates that the dilated vessels contain blood.

Complications.—Inflammations of the skin and lymphorrhœa are common when the lymphatics of the skin and subcutaneous tissues are involved. A scratch or an insignificant injury may rupture a lymph-vesicle, from which is discharged large quantities of lymph. [" In one case of lymphangiectasis involving the labia majora in which a fistula developed, Nieden found that in four hours there was an escape of one and a half liters of a milky, slightly yellowish liquid containing fat and resembling chyle."—Tillmanns' " Text-book of Surgery," Vol. I, p. 544.] The discharge of lymph may continue for days and weeks without impairing the general condition of the patient. It, however, macerates the skin which is continually bathed by it, and provides infection atria for phlegmonous inflammations and erysipelas. A lymphorrhœa is frequently followed by lymphangitis.

Treatment.—The treatment of the less extensive lymphangiectases limited to the skin and subcutaneous tissues is the same as that employed for lymphangioma. The dilated lymphatic vessels should be excised. Extensive swellings subside gradually under the pressure of well-applied bandages and elevation of the extremity. If there is a lymphorrhœa, dressings of oxid of zinc ointment should be applied to protect the surrounding skin. Lymph fistulæ frequently close after repeated cauterizations with silver nitrate. In the more resistant cases incision and tamponing of the wound with iodoform gauze is often necessary.

LITERATURE.—*Fr. Fischer.* Krankheiten der Lymphgefässe, Lymphdrüsen und Blutgefässe. Deutsche Chir., 1901.

CHAPTER V

DISEASES OF THE PERIPHERAL NERVES

(a) NEURALGIA

Definition—True and Symptomatic Neuralgia.—By neuralgia is understood a disease of the sensory nerves, the chief symptom of which is pain. It may occur as an independent affection (true neuralgia) or be merely symptomatic (symptomatic or secondary neuralgia) of some local or general lesion influencing the nerves. When occurring as an independent affection, no pathological changes are found in the nerves.

Characteristics and Symptoms.—The most important characteristics of neuralgia are intermittent or remittent attacks of severe, often agonizing, pain which radiates along nerves, nerve trunks, or plexuses, and subsides completely or incompletely after lasting for a few minutes or hours. The pain may recur upon the slightest provocation, such as pressure upon the nerve or movement on the part of the patient.

The symptoms which usually develop in middle life either begin suddenly, reaching their maximum intensity early, or are mild in the beginning and gradually increase in severity. Sometimes the pain begins without warning, at other times there are prodromata, such as mild shooting pain and tingling sensations. A few hours or days may intervene between the attacks. Frequently, when the attack is at its height, the pain is no longer limited to the nerve primarily involved, but radiates along adjacent nerve trunks (irradiation).

Neuralgia may extend over days, weeks, months, or years. The symptoms may disappear and not recur, or they may extend to other branches of the same nerve or plexus. Sometimes, especially in tri-

facial neuralgia, the patients are scarcely free from pain, which is so severe that they have often committed suicide.

A number of disturbances, of which the following are the most pronounced and frequent, follow interference with nerve function: (1) Sensory disturbances (the area supplied by a diseased sensory nerve is often hyperæsthetic, more rarely anæsthetic); (2) increase of glandular secretion (epiphora, increased flow of saliva and sweat); (3) blanching and flushing of the skin, depending upon the condition of the vessels; (4) reflex fibrillary muscular twitchings during an attack (e. g., twitching of the facial muscles in trifacial neuralgia); (5) trophic disturbances, such as atrophy of the skin, falling out of the hair, tendency to eczema, and the development of herpes (herpes zoster in intercostal neuralgia).

The general condition of the patient suffers when the neuralgia lasts for any length of time. Pain deprives the patient of sleep, and in trifacial neuralgia the taking of food is interfered with, as the movements of the jaws frequently incite attacks of pain. Psychic changes (irritability, melancholia) develop in the protracted and severe cases.

Causes of Neuralgia.—The causes of neuralgia are general and local. Among the general causes are a neuropathic temperament, exhausting physical labor, mental worry—all of which are frequently associated with strong emotions and sexual excesses, lessened bodily resistance, general weakness, and chronic constipation (in trifacial neuralgia, Gussenbauer); infectious diseases (malaria, typhoid fever, smallpox, influenza); and toxic agents, such as lead, copper, mercury, alcohol, and nicotin. Neuralgia also occurs in diabetes mellitus, being secondary to the changes in metabolism. The local causes are chilling of the part involved; crushing and laceration of nerve trunks; pressure by penetrating foreign bodies; traction and pressure exerted by scar tissue upon the surrounding nerves; pressure upon nerve trunks by displaced fragments of bone, aneurysms, varicose veins, gummas, and tumors (also amputation neuromas); and, finally, inflammation about nerve endings or trunks, such as periostitis of the mandible, suppuration of the accessory sinuses of the nose, carious teeth, ulcers of the mucous membranes, tuberculosis of the vertebra, sacrum, and ribs.

Neuralgialike pains may also be associated with tumors and diseases of the central nervous system (tumors at the base of the brain and of the spinal cord, tabes dorsalis, syphilitic meningitis, multiple sclerosis) (Oppenheim).

Nerves Most Commonly Involved—Pain Points and Diagnosis.—Neuralgia of the trigeminal nerve is the most common. Then follow in order of frequency, neuralgia of the sciatic, intercostal, and occipital nerves, of the nerves of the lumbar, pudendo-hæmorrhoidal and coc-

cygeal plexuses, and of the extremities. The peculiarities in the onset, symptoms, and clinical course of each of these different forms belong to the province of special surgery and nervous diseases.

In making a diagnosis of neuralgia it is important to note that the pain corresponds to the anatomical distribution of the nerve involved, and that it extends beyond the areas supplied by the nerve primarily involved only at the height of the attack. Certain points, the so-called *pain points*, are very sensitive to pressure, and an attack may be provoked by making pressure at these points. Such pain points are found where the nerve trunks or branches leave a bony canal, or where they can be easily pressed against some resistant band; for example, in neuralgia of the sciatic nerve at the border of the gluteus maximus muscle, in the middle of the popliteal fossa and below the head of the fibula, in neuralgia of the supraorbital nerve at the supraorbital notch, and of the infraorbital nerve at the corresponding foramen.

The local or general causes of the neuralgia should always be looked for, as the treatment which should be instituted depends upon the cause. A good example of this is malarial neuralgia (which subsides under quinin, or sciatic neuralgia (which often gives a clew to a pelvic tumor, or tuberculosis of the spine [perhaps still curable], or a tumor of the rectum). Bilateral neuralgias involving symmetrical nerves always suggest some central lesion, such as a tumor of the skull or of the base of brain; an intercostal neuralgia suggests a tumor of the cord or a tuberculous spondylitis. The symptoms may be unilateral in these lesions, and then the diagnosis is difficult if there are no other symptoms (cf. psamomma of the dura illustrated in Chapter VI, Part II, which caused a trifacial neuralgia, the ganglia being extirpated under the wrong diagnosis). Neuralgia may be mistaken for the false neuralgia occurring in hysterical patients, attacks in whom are generally induced by some psychic disturbance. It may also be mistaken for neuritis when a careful examination is not made.

Treatment.—The treatment of neuralgia is generally successful if the cause can be removed. This is less difficult when the cause is local and peripheral than when it is general or central. As a rule, the neuralgia disappears when scar tissue pressing upon the nerves, the tumor, fragment of bone, foreign body, etc., is removed and the inflammation subsides. General diseases should receive appropriate treatment, a hygienic mode of life should be adopted, the general condition of the patient improved, and constipation corrected.

If no cause can be found, or if the general or local cause has been removed without success, the remedies and procedures used in internal medicine should be tried (*vide* Edinger). Only the most important of these will be mentioned. Quinin (not in malarial neuralgia only),

arsenic in the form of Fowler's solution, potassium bromid, aconite, aspirin, and pyramidon are especially to be recommended. Local applications of heat (warm compresses, poultices, hot-water bags and bottles) and electricity, both the galvanic and faradic current, may be tried. Sometimes weak solutions of cocain, eucain, or Schleich's solution are injected about the affected nerve trunks to produce transitory anæsthesia or a one per cent solution of osmic acid is injected into the nerve to produce a degeneration of its fibers. Injections of alcohol along the tract of the nerve have recently been employed with some success. In severe cases morphin is required, but operative treatment should be instituted before the patient becomes accustomed to large doses and contracts the morphin habit.

If all these different methods have been tried without success, or if the general condition of the patient rapidly becomes worse, as the attacks recur more frequently, surgical treatment is indicated. Results can be promised only when there is no central cause, for in the latter case the pain persists even after removal of the nerve.

Neurotomy, Neurectomy, and Nerve Stretching.—Neurectomy (introduced by Abernethy, 1793) soon replaced *neurotomy* (first performed by Schlichting in 1748). The results following the latter were only temporary, as the sensory nerves rapidly regenerated. Even after neurectomy there is danger of the continuity of the nerve being reëstablished unless long pieces are removed. Nerve extraction devised by Thiersch (1889) is more reliable than either of the methods above mentioned, and should be tried in trifacial neuralgia before the removal of the ganglion is considered. In this method the nerve trunk is exposed at a suitable point, then grasped with forceps and twisted until all its connections are gradually torn. If patience is exercised, sections of the nerve from 10 to 20 cm. in length may easily be removed.

Even after extraction recurrences are frequent, especially in trifacial neuralgia. These recurrences are due to the regeneration of the nerve from central fibers which were not accessible when the nerve extraction was performed. Hartley and Krause (1892) performed a more radical operation in cases of this character, removing the Gasserian ganglion (literature by Lexer and Türk). Shortly before this Horsley had cut the sensory root behind the ganglion.

In the treatment of persistent neuralgias of the mixed nerves supplying the trunk and extremities, it may be necessary to perform a laminectomy, and after incising the dura mater to resect the posterior or sensory roots of the nerves involved (Chipault and others). In cases in which there are also muscular spasms it is not necessary to open the dura mater, as both roots may be resected where they join to form the nerve before it divides into its anterior and posterior divisions (cf. Schede).

Nerve stretching was first practiced by Billroth in 1869, then by Nussbaum in 1872, and was especially recommended by the latter.

It was first employed for the treatment of epileptiform attacks following contusions of nerves. Gärtner (1872) was the first to employ this method for the treatment of neuralgia involving the brachial plexus. It was soon tried in a number of different diseases; for example, the sciatic nerves were stretched for the relief of tabes dorsalis (Langenbuch), the facial nerve for convulsive tic, the spinal accessory for convulsive wryneck, the fifth cranial nerve for trifacial neuralgia. Finally the nerves were even stretched in tetanus (Vogt and others). The results which had been expected were not obtained, and the method was finally abandoned.

According to Schede, however, nerve stretching is to be recommended not only in the treatment of resistant neuralgias of mixed nerves and painful muscular spasms, but also in the treatment of nerve changes following neuritis. While a complete and permanent cure cannot be promised, considerable improvement may be expected. In spinal affections it has no effect upon the course of the disease, and in the treatment of neuralgias of sensory nerves, nerve extraction has been used in its place (Schede) for a long time.

In performing the operation of nerve stretching the nerve is exposed at the point desired, is isolated by blunt dissection, is then grasped between the thumb and index finger or by suitable tractors, and stretched both ways until it has been plainly lengthened.

The benefits derived from nerve stretching are due to the lessened conductivity following the trauma, to the degenerative and regenerative changes occurring in the nerve following the operation, and to the separation of the nerve from the cicatricial tissue which may surround it.

The best results have been obtained in the treatment of sciatica. Bloodless stretching of the sciatic nerve has also been attempted. In the latter procedure the patient is anæsthetized and the straightened extremity is flexed at the hip joint until the leg comes in contact with the face. The extremity is then maintained in this position for five minutes.

(b) NEURITIS

Definition.—Neuritis is an acute or chronic, serous or seropurulent inflammation of the perineurium and interstitial tissue of nerves, resulting in the degeneration of the fibers with secondary proliferation of the connective tissues of the nerve. The part of the nerve affected presents a fusiform swelling and is reddened, as a result of the inflammatory infiltration and hyperæmia. After the development and induration of

new connective tissue the nerve becomes hard, irregular, and nodular, and firmly adherent to adjacent structures.

Etiology.—The most common causes of neuritis are injuries of various kinds, and toxæmias associated with infectious diseases, chronic poisonings, and constitutional diseases. The most common injuries affecting nerves are lacerations and contusions, repeated blows or long-continued pressure on nerve trunks received in certain occupations (occupation neuritis) or while using a crutch (crutch palsy), the pressure of fragments of fractured bones and ends of dislocated bones, foreign bodies (such as a fragment of glass, point of a knife, a bullet), tumors, cervical ribs, etc. In open injuries involving the nerves and in cases in which pyogenic, tuberculous, or gummatous lesions have extended from bones or joints and have involved the nerves, bacterio-toxic and mechanical causes are combined. Neuritis may develop during the course of or subsequent to a number of infectious diseases, such as general pyogenic infections, especially of puerperal origin, typhoid fever, diphtheria, syphilis, etc., and in chronic poisoning due to lead, arsenic, alcohol, and nicotin. Cold, rheumatism, gout, diabetes mellitus, leukæmia, and arteriosclerosis are also to be considered as causes.

The inflammation following local causes gradually extends in the form of an ascending and descending neuritis toward the cord and periphery. When the cause is general, not infrequently a number of different nerves are involved (polyneuritis). In chronic alcoholism, lead and arsenic poisoning, paralysis of the extensors of the hands and feet, more rarely of the flexors, develops, while in diphtheria (*vide* p. 351) any nerve may be attacked.

Symptoms.—Acute neuritis may begin with chills and fever. Pain of a boring, tearing character, localized in the nerve primarily affected, which is increased by movements and pressure, is the most important symptom. Symptoms of irritation of the sensory and motor nerves consisting of paræsthesia, hyperæsthesia, and contractures follow the pain. As the lesion advances the nerves no longer conduct impulses, and then the reflexes disappear; anæsthesias, trophic disturbances, palsies, and later flaccid paralyses with muscular atrophy and the reaction of degeneration develop.

Chronic neuritis, unless it develops from an acute form, begins more insidiously. The pain in chronic neuritis is less severe, and nodular thickening may develop along the nerve involved (neuritis nodosa).

Acute cases of neuritis may subside after a few weeks, the nerve fibers regenerating. In chronic neuritis, functional disturbances which resist treatment or become permanent develop more frequently.

Diagnosis.—It is not always possible to make a diagnosis between neuritis and neuralgia. The character of the pain is of diagnostic value.

In neuritis it is continuous, while in neuralgia it occurs at intervals. In neuralgia the tenderness is limited to certain points (pain points, p. 695), while in neuritis the pain extends along the entire nerve, which is often perceptibly thickened. Finally the marked sensory, motor, and trophic disturbances which develop rapidly in neuritis are wanting in neuralgia. It may be difficult at times to differentiate multiple fibromas of nerve trunks from the nodular form of neuritis. The former, however, are usually associated with soft fibromas of the skin and pigmented areas, and besides there is no interference with nerve conduction.

Treatment.—In the treatment an attempt should be made to remove the mechanical or inflammatory cause, and then to immobilize the extremity involved. Morphin, sodium salicylate, salol, and aspirin are the drugs usually recommended. [Morphin should be used sparingly, however, as there is always the danger that the patient may contract the habit.] In chronic cases an attempt should be made to favor regenerative processes by massage, electricity and baths. Contractures and paralyses should receive appropriate treatment.

If an attempt is made to remove a local cause, such as a foreign body or cicatricial tissue, the nerve should be exposed for some distance, the adhesions between adjacent tissues and the perineurium dissected away, and the nerve stretched. Nerve stretching in chronic cases has a favorable action. It not only frees the nerve from adhesions, but stimulates regenerative processes.

LITERATURE.—*Chipault* et *Demoulin.* La résection intradurale des racines médullaires post. Gaz. des hôpitaux, 1895, No. 95.—*Edinger.* Behandlung der Neuralgie. Handb. der Therapie von Penzoldt u. Stintzing, Bd. 5, Part II, p. 553.—*Th. Kölliker.* Die Verletzungen und chir. Erkrankungen der periph. Nerven. Deutsche Chir., 1890.—*Fedor Krause.* Die Neuralgie des Trigeminus. Leipzig, 1896.—*Lexer.* Zur Operation des Ganglion Gasseri nach Erfahrungen an 15 Fällen nebst Zusammenstellung der ausgeführten Ganglionexstirpationen von W. Türk. Arch. f. klin. Chir., Bd. 65, 1902, p. 843.—*Oppenheim.* Lehrbuch der Nervenkrankheiten. Berlin, 1904.—*Schede.* Chirurgie der peripheren Nerven und des Rückenmarkes. Handb. d. Therap. von Penzoldt u. Stintzing, Bd. 5, Part II, p. 738.—*Thiersch.* Ueber Extraktion von Neryen. Chir. Kongr.-Verhandl., 1889, I, p. 44.

CHAPTER VI

DISEASES OF JOINTS

(a) DISLOCATIONS AND SUBLUXATIONS

DISLOCATIONS and subluxations may be *congenital* or *acquired.* Of congenital dislocations, those of the hip are the most frequent. This malformation is more frequently unilateral than bilateral, and is more

common in girls than in boys. Congenital dislocations of the shoulder, knee, and elbow joints are infrequent, as are also those of the head of the radius, of the external malleolus, of the wrist, of the fingers, of the patella and clavicle.

Congenital Dislocation.—*Theories as to Causes.*—The causes of congenital, frequently also of acquired dislocations, are not clear. There are a number of theories as to the cause of congenital dislocation of the hip. [It is probably due in some cases to malposition of the fœtus in the uterus, or to some irregularity in the shape of the uterus.] If in the beginning of pregnancy there is an insufficient amount of amniotic fluid, the walls of the uterus will be closely applied to the fœtus, its thighs will be forcibly flexed and adducted, and the head of the femur will be forced out of the acetabulum (Hoffa). Abnormal amniotic bands may interfere with the normal development of any of the other joints.

Symptoms and Signs of Congenital Dislocations.—The deformity resulting from a congenital dislocation may be more or less marked at birth. The symptoms, however, as is usually the case in congenital dislocations of the hip, may not be noted until the child begins to walk.

[Patients with a congenital dislocation of the hip have a peculiar waddling gait, which becomes very pronounced when but one side is affected.] The signs common to the acquired are found in congenital dislocations, but the head of the femur is usually freely movable, and movements cause no pain. In fat children the head of the femur cannot be palpated unless there is considerable displacement. X-ray pictures should be taken. The most accurate diagnosis can be made in this way.

Reduction of Dislocation.—Reduction of the dislocation is, as a rule, difficult only in the old cases with contracted soft tissues, and in those cases with secondary joint changes resembling those of arthritis deformans. After reduction, the parts must be maintained in position for a long time by properly applied bandages. If the dislocation cannot be reduced after the shortened muscles have been stretched and lengthened, an operation in which the joint is exposed and the obstacles to reduction are removed should be performed.

Prognosis.—Usually the functional results following bloodless reduction are better than those obtained by operative procedures. The latter should be employed only in bad cases, after attempts at reduction by the bloodless method have failed. There is always danger of anchylosis after reduction by the open method.

Acquired Dislocation—Pathological and Traumatic.—Acquired dislocations which are secondary to some disease of the joints are known as

spontaneous or pathological dislocations, and are differentiated from the traumatic, which are produced by force.

Causes.—Pathological dislocations may follow inflammation of the joints in which the capsule is distended by a large exudate or the articular surfaces, forming the joint, and the capsule are destroyed. Dislocations following distention of the capsule are known as *distention dislocations*; those following destructive changes in the joint as *destruction dislocations.*

Pathological dislocations may follow deformities due to defects in or shortening of neighboring bones; for example, dislocation of the head of the ulna after resection or pathological shortening of the radius, outward and upward dislocation of the head of the fibula after necrosis of the tibia. Dislocations frequently follow paralysis of the muscles surrounding a joint. If all the muscles are paralyzed, the weight of the entire extremity is thrown upon the capsule of the joint, which becomes stretched and lax. The articular surfaces then fall away from each other and a paralytic flail joint develops. A dislocation of this kind may be easily reduced, but recurs as soon as the pressure exerted by the hands or some special apparatus is removed. The diagnosis of a dislocation of this character is easily made, as the soft tissues are atrophic and the exact positions of the articular ends of the bone can easily be determined. A dislocation may occur, even if only a few of the muscles surrounding a joint are paralyzed, as the antagonistic muscles which are not paralyzed gradually separate the articular ends of the bone and but little force is required to complete the dislocation. For example, when the abductors and rotators of the thigh are paralyzed, the adductors produce a dislocation backward. If the conditions are reversed a dislocation forward occurs.

Symptoms and Diagnosis.—The symptoms and functional disturbances following pathological dislocations differ, depending upon the cause. The diagnosis is based upon the abnormal position of the articular ends of the bones and upon the symptoms of the disease of the joints.

Treatment.—Reduction of the dislocation and treatment of the accompanying inflammation of the joint or bones should be combined. If the dislocation follows inflammation of the joint or of the articular ends of the bones, the reduction should be made by gradual extension rather than by the manipulations used in the reduction of traumatic dislocations. If the dislocation is not recent, it may be necessary to reduce it by the open method. Paralytic contractures should receive appropriate treatment. Resection of the ends of the bones is indicated when there is anchylosis and in destruction dislocations. The tendons should be shortened and some apparatus worn after the reduction of paralytic dislocations.

(b) CONTRACTURES AND ANCHYLOSIS

By contracture, strictly speaking, is understood the results of muscular contraction—that is, the approximation of two neighboring parts of the body. By the term as it is employed to-day is understood, however, not only the condition produced by active muscular contractions, but also the faulty positions in which joints may become more or less fixed as the result of the contraction and shortening of the soft tissues surrounding them or by the permanent contraction of a group of muscles.

Depending upon the position in which the joints become fixed, contractures in the position of flexion, extension, adduction, abduction, rotation, pronation, and supination are described. If the contracture is marked (most often in flexion) an acute angle may be formed between the approximated parts; if less marked, an obtuse angle.

The joint surfaces may maintain their anatomical relations or be partially or completely separated. In the former case they may be united by fibrous bands or masses of bone (fibrous or bony anchylosis).

Congenital Contractures.—Some congenital contractures are due to failures of development; they are then frequently associated with bony defects. Some are due to pressure of the uterus upon the fœtus, when there is insufficient amniotic fluid, or to constriction by the cord and amniotic bands. They occur occasionally as flexion contractures of the hip, of the knee, and of the wrist, and not infrequently of the little finger. Congenital contractures occur frequently in the foot in the form of different varieties of club-foot. Talipes varus is most common, while flat-foot (by pronation), talipes equinus, and calcaneus are rarer. Congenital spastic contractures due to congenital defects of the nervous system or to injuries of the cerebrum during birth also occur.

In all congenital contractures malpositions develop as the muscles, fascia, and ligaments contract and the mobility of the joints is decreased.

Acquired contractures are of *dermatogenous, desmogenous, myogenous, neurogenous,* and *arthrogenous* origin (Hoffa).

Dermatogenous Contractures.—Dermatogenous contractures are produced by scars in the skin, and may be overcome by excising the scar and uniting the edges of the resulting defect or covering it with skin grafts.

Desmogenous Contractures.—Desmogenous contractures are produced by deep scars (for example, after burns, necrosis of tendons and fascia following suppuration and injuries), or in the hand by overgrowth and contraction of the palmar fascia. In the latter the contracture begins, and is most pronounced in the ring and little fingers. As the contrac-

tures advance the fingers involved become flexed (Dupuytren's contracture). The thumb is more rarely involved.

Excision of the scar tissue, in Dupuytren's contracture of the altered palmar fascia, is the most efficient treatment. The portion of the skin which is sometimes involved in the scarlike tissue should also be excised. After removal of the scar and correction of the contracture, the defect should be covered with pedunculated skin flaps or skin grafts (Lexer). It is most difficult to correct contractures following phlegmonous inflammation of the tendon sheaths (tendogenous contractures). When the tendons are destroyed forcible extension with immobilization in the corrected position, and even excision of the scar with skin grafting, freeing of the tendons from surrounding scar tissue, and lengthening of the same give but temporary results. Adhesions soon form again, and the prognosis as regards function is hopeless.

Myogenous Contractures.—Myogenous contractures are due to shortening of the muscle fibers, which may be the result of certain forms of atrophy, of injury, and of inflammation of muscles. If an extremity is held in one position for a long time, the points of origin and insertion of the muscles become approximated, and finally the muscles become adapted to their new conditions. These contractures develop in certain occupations (habit contractures with flexion of the fingers in cabmen and handicraftsmen, adduction and flexion of the thigh in patients confined to bed for a long time), when an extremity is intentionally held in a certain position (for, example, when the foot of a shortened extremity is held in the position of talipes equinus), and finally when an extremity is immobilized in a definite position for a long time (pronation and supination, flexion of the forearm, flexion of the foot, flexion of the thigh when elevated after amputation) or when the bed clothes are allowed to exert pressure for some time upon the anterior part of the feet of very sick patients. The weight of the foot also contributes to the development of the last type of contractures.

Myogenous contractures are frequently caused by diseases and inflammation of the muscles. In the beginning the muscles involved are contracted and held rigid, as extension causes pain. The best-known example of this condition is the so-called rheumatic wryneck, which often quickly subsides after massage. In suppurative inflammation, in tuberculosis and syphilis of the muscles, in fibrous myositis, in ischæmia, and inflammation following contusions and lacerations of the muscle fibers cicatricial tissue forms, which later contracts, producing distressing and often unsightly deformities. The best known of these are contractures of the fingers after phlegmonous inflammation of the muscles upon the anterior surface of the forearm, flexion contracture of the thigh in tuberculous spondylitis following cicatricial contraction of the

ilio-psoas muscle, claw hand after ischæmic paralysis of the muscles of the forearm (Fig. 243), cicatricial wryneck following laceration of the sterno-cleido-mastoid muscle during labor or secondary to fibrous myositis.

In the milder cases marked improvement follows massage and passive motion. In the severer cases, if results are not obtained by forcible correction under general anæsthesia, division, lengthening and transplantation of tendons may be tried. Sometimes relief follows resection of the bones. [A number of cases have been reported lately in which ischæmic contractures have been greatly improved by resecting the bones. Resection of the bones of course produces a relative lengthening of the shortened tendons.] When muscles are inflamed an attempt should be made to prevent contractures by dressing and maintaining the parts involved in a correct position. According to Hildebrand an attempt should be made in ischæmic contractures to dissect the nerves free from scar tissue and to place them where they will no longer be compressed by it (*vide* p. 656).

Neurogenous Contractures.—Neurogenous contractures are often accompanied by shortening of the muscles, but the principal lesion is in the nervous system. *Reflex*, *spastic*, and *paralytic* forms of neurogenous contractures are described. Reflex contractures, due to irritation of sensory nerves, may occur in almost any painful lesion, and are frequently the first symptom. In arthritis the joint assumes the least painful position, and is maintained in it by muscular contraction. In order to prevent pain the anterior abdominal wall becomes boardlike and is held rigid, and abdominal respiration is suspended in the beginning of acute peritonitis; the head is held rigid in acute suppurative inflammation involving the side of the neck; and the jaws are held closed when phlegmonous inflammation attacks the muscles of mastication or the tissues surrounding them. A painful, immobile flat-foot is an example of a reflex contracture. Sometimes foreign bodies situated upon nerves or scar tissue pressing upon them cause changes which result in contractures.

These contractures usually subside when the cause is removed. If the *reflex* contracture continues for some time, the muscles become shortened and contracted, and the treatment described above for myogenous contractures must then be employed. Pure reflex contractures may be easily corrected under anæsthesia. Recurrences should be prevented by immobilizing the parts in a proper position.

The *spastic* forms are due to abnormal innervation or to a pathological irritation of a motor nerve (Hoffa). They may be unilateral or bilateral and are almost always of central origin—that is, they follow a number of different lesions of the brain and cord (cerebral tumors and

hæmorrhages, multiple sclerosis, hydrocephalus, compression myelitis, sclerosis of the cord, chronic meningitis, hysteria, etc.). Congenital spastic contractures are due to defects or birth injuries of the cerebrum. [Spastic contractures of the fingers, known as writer's cramp, which occurs in bookkeepers and stenographers, is a neurosis.] The muscles are in a state of pathological contraction, but are weak, and for this reason a spastic is differentiated from a flaccid paralysis. The muscles feel hard and rigid, can be extended only with difficulty by passive motion, and return to the contracted position as soon as the pressure is released. The tendon reflexes are exaggerated. In congenital spastic contractures frequently only the legs are involved, and especially the flexor and adductor muscles. As a result of the talipes equinus, of flexion of the knee and hip joints and adduction of the thigh, the gait is awkward. The adductors become stronger than the abductors, and a peculiar, characteristic, cross-legged gait is produced. Whenever an attempt is made at walking a number of other muscle groups are thrown into action. In severe cases walking is impossible.

Mechanical treatment (massage, extension of the muscles by passive motion, and extension apparatus), often combined with division of the tendons of the muscles most involved with subsequent immobilization in plaster-of-Paris dressings, is indicated. Resection of the motor nerves has also been recommended.

Paralytic contractures are most frequently the result of anterior poliomyelitis, of injuries of the peripheral nerves, of neuritis, and of different lesions of the brain and spinal cord. These contractures following flaccid paralysis of a single muscle or group of muscles are due to the contraction, and later, if not used, to the shortening of the antagonistic non-paralyzed muscle or group of muscles (Seeligmüller's antagonistic-mechanical theory) or to the weight of the extremity, which is thrown upon the joints. Sometimes the intact muscles hypertrophy. Paralytic talipes equinus develops after paralysis of the extensor group of muscles, and is due to the contraction of the muscles of the calf and to the weight of the foot. The weight of the foot may, on the other hand, be sufficient to prevent the development of a talipes calcaneus, which may follow a paralysis of the flexor groups of muscles.

Etiology, Pathology, and Symptoms of Anterior Poliomyelitis.— The etiology of infantile paralysis (poliomyelitis anterior) is not exactly clear. [" The onset is usually sudden, and the paralysis may occur before the development of the general symptoms. The legs are more frequently involved than the arms; the muscles are usually affected in functionally similar groups, such as the flexors of the forearm, and then very rapidly begin to undergo contractures. These produce deformities, particularly various forms of club-foot, scoliosis or lordosis,

and contractures of the hand.''—Musser, '' Medical Diagnosis,'' p. 1038.] There are no sensory, bladder, or rectal disturbances; the reflexes are abolished or weakened. The pathological changes, consisting of degeneration of the anterior horn cells with subsequent degeneration of the fibers arising from them and the muscles supplied by them, are most marked in the cervical and lumbar enlargements of the cord.

Massage, electricity (galvanic), active and passive motion, warm baths, and inunctions are indicated in the treatment of paralytic contractures. Supporting apparatus or immobilizing dressings should be applied to maintain the parts in correct position. Elastic bands, by the contraction of which the absence of muscular action is partially compensated, may be attached to the mechanical apparatus.

In some cases tendons may be transplanted to advantage. The malformations due to shrinkage and contraction of antagonistic muscles may be relieved by tenotomy. If the paralysis is extensive a mechanical support must be worn or the joint opened, the articular surfaces removed, and an attempt made to secure a bony anchylosis (arthrodesis) in a good, useful position. The results of the latter operation are, however, always doubtful, as the callus formation may be insufficient and the union poor. Frequently a good position of the parts may be obtained by shortening the tendons involved.

Arthrogenous Contractures.—Arthrogenous contractures following injuries and inflammation of joints are due to shrinkage and contraction of the soft tissues, such as the synovial membrane, ligaments, and periarticular tissues, entering into the formation of the joint. In inflammations of joints the contractures are reflex and myogenous at first, but later become arthrogenous when the capsule contracts. Long-continued immobilization after injuries favors the development of contractures.

The stiffness of the joint or anchylosis develops in different ways. Contraction of the capsule alone may interfere with the movements of the joint, as in all forms of contractures connective tissue develops in the joint which is no longer used, uniting the articular cartilages, and if these are destroyed, the exposed surfaces of the bones. These fibrous adhesions (anchylosis fibrosa intercartilaginea) later become ossified, and bony anchylosis (anchylosis ossea) develops. Bony anchylosis may develop without a preceding fibrous anchylosis, when the articular cartilages are destroyed or when callus forms within the joint after fractures involving the articular ends of bone.

The treatment of arthrogenous contractures depends upon the degree of anchylosis. In fibrous and capsular anchylosis the deformity may be corrected or improved by gradual extension by weight and pulley. Passive motion may often be combined with gradual extension to ad-

vantage. If the lighting up of an old inflammatory process is not to be feared—for example, in traumatic anchylosis—the deformity may be forcibly corrected under general anæsthesia.

When there is a firm fibrous, cartilaginous, or bony anchylosis, resection of the joint is generally indicated, the object being the formation of a movable joint or one fixed in a position which will be useful.

In resection of the knee and ankle joint an attempt should be made to obtain a bony anchylosis in a useful position; in resection of the shoulder and elbow an attempt should be made to secure movement by instituting early active and passive motion. [Murphy has recently obtained some brilliant results in cases of bony anchylosis of the elbow and knee by an operation called arthroplasty, in which, after resecting the anchylosed joint, a movable joint is obtained by placing between the bones a flap of connective tissue and fat from which a new synovial membrane is formed, securing a movable joint.] If, as is frequently the case in pathological dislocations, the adhesions are very extensive, osteotomy below the line of the former joint is generally to be preferred to resection. If the bone is cut through obliquely (König and Hoffa) the deformity cannot only be corrected, but the shortening can also be overcome by making traction during the process of repair by weight and pulley.

Tenotomy, lengthening, shortening, and transplantation of tendons are the operations most frequently employed for the correction of myogenous contractures.

Technic of Tenotomy.—Tenotomy or cutting the tendons of the shortened muscles may be performed by the subcutaneous or open method. In the subcutaneous operation a narrow-bladed knife or Dieffenbach's sickle-shaped tenotome is used. [Tenotomy is discussed in Bryant's "Operative Surgery," Vol. I, pp. 329 and 330, as follows: "Tenotomy consists in making a subcutaneous or open division of a tendon for the purpose of overcoming or alleviating a deformity usually due to muscular contraction. Since the advent of antiseptic surgery, open division can be practiced with comparative uniformity if a rigid adherence to its tenets be maintained.

"However, it is wiser to hold to the subcutaneous method than to invite unnecessarily the mishaps that may follow a faulty technic in the open one. In order to practice tenotomy successfully the exact location of the offending structure should be determined, together with the important contiguous vessels, nerves, etc. Many of the large tendons are easily located by their natural prominence. Others that ordinarily lie concealed become apparent if contraction and deformity have occurred, and still more conspicuous if placed upon the stretch by

the surgeon. The principles governing tenotomy should be well considered before a tendon is divided, otherwise an expedient of great good may become mischievous and even destructive in its results.

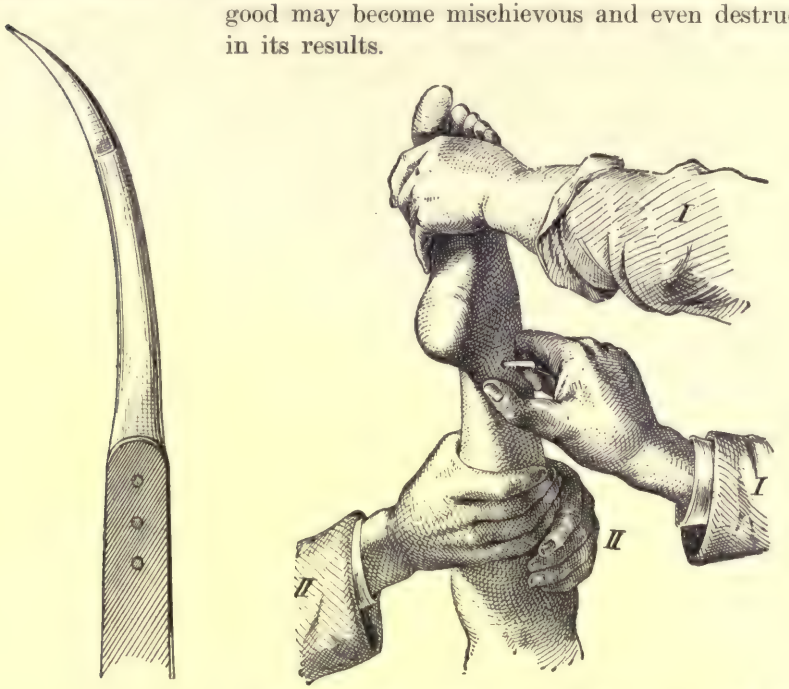


FIG. 263.—DIEFFENBACH'S TENOTOME AND SUBCUTANEOUS TENOTOMY OF THE TENDO ACHILLIS.

"The operation of tenotomy is simplified by attention to the following order of procedure:

"1. Secure complete aseptic technic.

"2. Indicate on the handle of the scalpel the direction of the cutting edge.

"3. Carefully note the length of the blade, so as to regulate the extent of the division of the tissues.

"4. Avoid, if possible, the division of a tendon as it passes through a special sheath.

"5. Divide the tendon at a point of greatest forced prominence, provided the division be consistent with the safety of important contiguous structures. If reflex spasm be provoked by 'point pressure,' the tendon should be divided at the point exhibiting the greatest reflex manifestation (Sayre).

"6. Make tense the structure to be divided, and so pinch up or push aside the skin at the point of proposed division that when the skin is relaxed the opening in it will not correspond to the divided tendon.

" 7. Insert the blade on the flat close to the surface of the tendon to be divided, turn the edge toward the tendon and carefully sever it with a guarded sawing motion, aided by pressing the tendon on the cutting surface of the knife. If incautious force be made, not only the tendon but the superimposed tissue may be divided, thus complicating the treatment and recovery.

" 8. Carry the edge of the blade away from important structures when possible.

" 9. Withdraw the blade while upon the flat. Follow the withdrawal with firm pressure upon the parts with the thumb which should finally rest on the incision. This act will press the blood and air from the wound, as well as prevent air from entering it. Close the wound with a horsehair stitch and seal it with antiseptic collodion. The application and confinement to the wound of an antiseptic pad is often quite sufficient for the requirements of healing.

" 10. Rectify the deformity and confine the part immovably until repair is well advanced."]

This operation, devised by Stromeyer, was very popular in Dieffenbach's time. At the present time it is almost never employed except for division of the tendo Achillis, of the tendons about the popliteal space, and the adductors. The last is really a myotenotomy, as the muscle fibers are also divided.

The open method, in which the tendon is divided after having been exposed by a free dissection, has a number of advantages: (1) The relations of the different structures can be seen, (2) injuries of the blood vessels may be avoided, (3) and shortened contracted bands of fascia as well as the affected tendon may be divided. In cicatricial wryneck the contracted fascia and muscle are both important factors in producing the deformity.

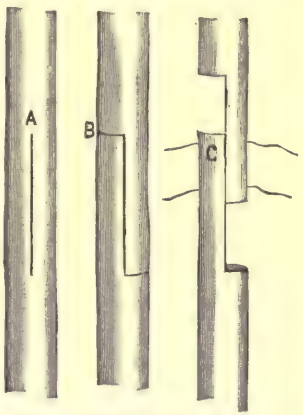


FIG. 264.—ANDERSON'S DOUBLE-FLAP METHOD. A, Longitudinal division; B, flaps formed; C, tendon lengthened, flap united. (Bryant's "Operative Surgery.")

Tenotomy throws a muscle out of action for a short time only. The blood clot forming between the divided ends soon becomes infiltrated with germinal tissue which is later transformed into a scar, and the continuity of the tendon or muscle is then reëstablished. The scar is comparable to the callus uniting fragments of a fractured bone. Passive motion should be begun at the end of a week, in order to maintain the lengthened condition of the tendon or muscle and to prevent the recur-

rence of shortening and contractures, which is frequent when proper after-treatment is not instituted.

Tenoplasty, Indications and Technic.—*Tendon lengthening* may be employed to correct the deformities resulting from the shortening of tendons or muscles. It is practiced most frequently upon the larger tendons, such as the tendo Achillis and the ligamentum patellæ. A tendon may be lengthened, after it has been exposed, by making alternate free incisions at its borders, with subsequent forcible extension. According to Boyer, the tendo Achillis may be lengthened by making two lateral incisions in it with the tenotome, one incision being just above the heel, the other somewhat higher on the other side of the tendon. [Bryant says that the making of alternate free incisions at the borders of a tendon—the accordion plan—so as to cause the tendon to assume an accordionlike appearance when lengthened is much more ingenious than practical.]

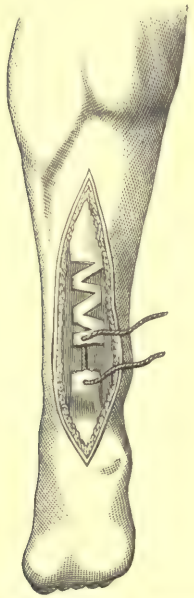


Fig. 265.—A. Poncet's Accordion Method. (Bryant's "Operative Surgery.")

Fig. 266.

Fig. 267.

Fig. 266.—Incision Method. (Bryant's "Operative Surgery.")

Fig. 267.—Tendon Lengthened in Incision Method. (Bryant's "Operative Surgery.")

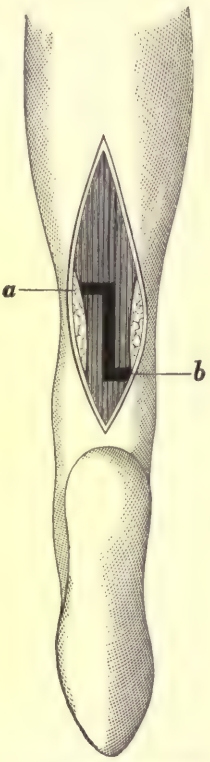


Fig. 268.—Lengthening Tendo Achillis. (Bryant's "Operative Surgery.")

Tendons may be lengthened and their continuity still be preserved by making a Z-shaped incision into the tendon and then making extension. When the tendon is extended the ends of the Z can be dis-

placed and sutured together. Tendon lengthening is used in place of tenotomy when the shortening is extreme, and in the treatment of contractures involving the tendons of the muscles of the fingers where subsequent failure of union of the divided ends is feared.

Tendon shortening is employed for the purpose of improving the action of muscles where power is lessened as the result of a complete or incomplete paralysis, and for the purpose of maintaining the joints in a correct or useful position. [Bryant shortly summarizes the general principles of tendon shortening as follows: "The removal of a proper segment of a tendon and union of the divided extremities can be accomplished by either a simple oblique incision or lateral apposition and union or by the introduction of the wedge-formed extremity of one into the split end of the other and fixation with sutures."]

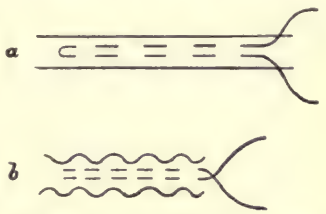


FIG. 269.—PLICATION OF A TENDON. (After Lange.)

Tendons may also be shortened without division by plication or folding upon a heavy silk suture. In paralytic flail joints, shortening of a number of the tendons of the muscles surrounding the joint (tendinous fixation) may be done to advantage.

Transplantation of Tendons.—The displacement or transplantation of tendons may be performed for the correction of paralytic contractures. This method, the description of which has already been given in discussing the repair of traumatic tendon defects, may be combined in a number of different ways with tendon lengthening and shortening. The following are simple examples of this most useful procedure: In paralytic club-foot the paralyzed peronei muscles may be divided and their distal ends united with a flap from the functionating tendo Achillis (*vide* p. 536); in paralytic flat-foot the tendon of the paralyzed tibialis anticus may be divided and its distal end united with the non-paralyzed extensor hallucis longus; in paralysis of the quadriceps extensor, F. Krause has separated the tendons of the flexors at their attachment and sutured them to the patella; in the case of paralysis of the musculospiral nerve Hoffa used a similar method, separating the flexor carpi radialis and ulnaris at their insertions and suturing them into the distal parts of the extensors.

In all cases the tendons must be united under such tension that the deformity will be overcorrected. If repair occurs, after immobilization in the overcorrected position for six or eight weeks, the results are usually very good, as the displaced or transplanted muscle or tendons assume the function of the paralyzed ones.

LITERATURE.—*Bardenheuer.* Ischämische Kontraktur. Festschrift zur Eröffnung der Akademie. Köln, 1904, p. 34.—*Bayer.* Eine Vereinfachung der plastichen Achillotomie. Zentralblatt für Chirurgie, 1901, p. 37.—*Drobnik.* Ueber die Behandlung der Kinderlähmung mit Funktionsteilung und Funktionsübertragung der Muskeln. Deutsche Zeitschr. f. Chir., Bd. 43, 1896, p. 473.—*Gerlach.* Klinisch-statistischer Beitrag zur Frage der Sehnenplastik und Sehnentransplantation. I.-D. Rostock, 1904.—*Hoffa.* Die Orthopädie im Dienste der Nervenheilkunde. Jena, 1900;—Lehrbuch der orthopädischen Chirurgie. Stuttgart, 1906;—Ueber Enderfolge der Sehnenplastik. Chir. Kongr.-Verhandl. Berlin, 1904, I, 24.—*Fedor Krause.* Ersatz des gelähmten Quadriceps femoris durch die Flexoren des Unterschenkels. Deutsche med. Wochenschr., 1902, p. 118.—*Nicoladoni.* Nachtrag zum Pes calcaneus und zur Transplantation der Peronealsehne. Arch. f. klin. Chir., Bd. 27, 1882, p. 660.—*Oppenheim.* Lehrbuch der Nervenkrankheiten. Berlin.—*Rosenkranz.* Ueber kongenitale Kontrakturen der oberen Extremitäten. Zeitschr. f. orthop. Chir., Bd. 14, 1905, p. 52.—*Vulpius.* Ueber die Heilung von Lähmungen und Lähmungsdeformitäten mittels Sehnenüberpflanzung. v. Volkmanns Samml. klin. Vortr. N. F., No. 197, 1897.

(c) SPECIAL DISEASES OF THE JOINTS

(1) CHRONIC SEROUS SYNOVITIS

Nature of Chronic Synovitis.—The symptoms of chronic irritation of the synovial membrane which leads to the formation of a serous exudate, thickening of the joint capsule, and hypertrophy of the synovial villi are similar to those of acute serous synovitis, with this difference, that there is little or no inflammatory reaction. Chronic synovitis is much more frequently the result or symptom of some other disease of the joint than an independent clinical entity. It follows rheumatism, hæmarthrosis, floating bodies, and arthritis deformans, or develops in a previously healthy joint as the first symptom of some specific disease, such as tuberculosis, syphilis, arthritis deformans, or neuropathic arthritis.

Usually, chronic serous synovitis develops in but one joint. The knee is affected most commonly, but it also occurs frequently in the elbow, ankle, and wrist joints. Involvement of many joints is rare and suggests some general cause, such as chronic articular rheumatism, articular syphilis, etc. The changes in the form of the joints are rather characteristic as the capsule is distended and prominent at all yielding points and the normal contour of the joint with its prominences and depressions is obliterated. Chronic serous synovitis develops slowly and may remain stationary for a long time. Exacerbations following use of the joint are frequent, but sometimes the synovitis subsides spontaneously when care is exercised. If the synovial fringes become hypertrophied and inflammatory masses form in the capsule, a condition resembling osteoarthritis develops. The sharp contour of the tense capsule then becomes lost and the palpable thickenings and nodules

in the inflamed capsule gradually become fused with the surrounding tissues.

Symptoms.—The symptoms in the beginning are insignificant. Unless the synovitis follows a hæmarthrosis or some acute painful inflammation, the patient is often unable to state exactly when the trouble began. A sense of fullness in the joint, of uselessness and weakness of the extremity is frequently the only complaint. Later in neglected cases when the ligaments have become relaxed malformations (such as genu valgum, g. varum, g. recurvatum, and subluxations) develop. Painful anchylosis develops if the capsule becomes thickened and the villi hypertrophied.

Diagnosis.—The diagnosis of chronic serous synovitis is not difficult. The chronic, almost painless, course, the changes in the form of the joint, the signs of fluid elicited by palpation and patellar balottement are characteristic. Hypertrophied synovial fringes and inflammatory masses in the capsule are most easily palpated after the exudate has been partially removed. Then, when the joint is moved, a crepitus and rubbing can be felt and heard.

It is frequently difficult to determine the cause of the synovitis when it is the first symptom and not the result of some previous disease of the joint. In these cases further observation is necessary before an exact diagnosis can be made.

Treatment.—If the exudate is small in amount, rest, elastic compression, massage, and passive motion are indicated. If the symptoms do not subside after this treatment, or if the effusion is great, the latter should be removed by aseptic puncture, with subsequent washing out of the joint with a three per cent solution of carbolic acid. After aspiration and injection the joint should be immobilized for a few days. Absorption of an exudate is favored by massage and hot-air treatment; active and passive motion prevent the formation of adhesions. Painful thickenings of the synovial membrane may require removal.

(2) CHRONIC ARTICULAR RHEUMATISM

Nature and Pathology.—By chronic articular rheumatism is understood a chronic, painful inflammation of the synovial membrane, capsular ligament, and periarticular tissues. Hypertrophy of the synovial membrane causes a swelling of the joint, contraction of the tissues of the capsule, limitation of motion.

Frequently the articular cartilages become fibrillated and destroyed, and replaced by a vascular connective tissue. In this way the joint cavity is gradually obliterated; anchylosis and contractures, to which atrophy of the muscles surrounding the joint and contraction of the capsule contribute, develop. The dry form (arthritis sicca) of chronic

articular rheumatism is more common than the form associated with a serous exudate and ending in hydrarthrosis. [" Chronic articular rheumatism never leads to suppuration and never to true caries, the pathological changes presenting more of a similarity to arthritis deformans, except that in the latter disease there is more of an increased growth of cartilage, while in the former the cartilage is replaced by vascular connective tissue. But deformities of the joints, subluxations and luxations develop in chronic articular rheumatism as they do in arthritis deformans."—Tillmanns' " Text-book of Surgery," Vol. I, p. 670.]

The nature of articular rheumatism is obscure. There are a number of transitional forms between chronic serous synovitis and chronic articular rheumatism, and between the latter and arthritis deformans. It is frequently mistaken for gout, arthritis deformans, gonorrheal arthritis, even for tuberculosis. It should also be remembered that a number of lesions which differ clinically and etiologically are grouped under the term chronic articular rheumatism, as characteristics which make a differential diagnosis possible are wanting.

Etiology.—Nothing definite is known concerning the etiology of this disease. It is questionable whether the short bacillus demonstrated by Schüller is to be accepted, and yet it cannot be doubted that at least some of the cases are due to bacterial infections. The not infrequent development of chronic articular rheumatism from the acute form, the similarity of the former to the arthritis of gonorrheal origin, the acute and subacute exacerbations, which are frequent during the chronic course of the disease, all indicate a bacterial origin. Clinical experience has demonstrated that getting wet, exposure to cold, and residence in damp, cold dwellings or regions favor the development of the disease.

The disease is observed almost exclusively in adults, the female being more frequently attacked than the male. Chlorosis seems to be an etiological factor in young girls, arteriosclerosis in old people.

Usually a number of different joints are involved, rarely a single one. In severe cases all the joints may be attacked. The disease is most common in the knee and shoulder joints and in the joints of the fingers and toes.

Symptoms and Course.—The onset is at times slow and insidious; at other times the disease develops as a sequela to acute articular rheumatism. Schüller has differentiated three forms—the *simple, severe,* and *anchylosing*—depending upon the clinical course of the disease and the pathological changes in the joints. In the simple form the pain in the joints, which gradually become swollen as the capsule thickens, is slight. It is increased by movements and pressure, is most marked in the morning after the night's rest, and when the patient attempts to

walk after sitting for a number of hours. The pains come and go. Exacerbations, accompanied by an effusion into the joint rendering motions more difficult, become frequent, while the swelling of the joints increases and becomes more distinct as the muscular atrophy increases and the stiff joints assume abnormal positions. The deformities are most marked in the hands. The metacarpo-phalangeal joints become very prominent upon the dorsum of the emaciated hand, the proximal phalanges become extended, while the remaining ones become flexed and the hand (on account of its weight) becomes displaced to the ulnar side (Fig. 270). Subluxations of the proximal and lateral displacements of the distal phalanges in extension are frequent. In the larger joints, where the capsule is accessible, the hypertrophied synovial fringes may be palpated as small nodules. During movements these masses rubbing upon each other produce a peculiar creaking and rubbing sensation. The symptoms and pathological changes remain stationary or pass into those of the severe form.

Fig. 270.—Chronic Arthritis of the Joints of the Fingers. (Woman fifty-five years of age.)

In the severe form the hypertrophy of the synovial villi is marked. In the course of time the entire surface of the synovial membrane becomes covered with simple, club-shaped or branched villuslike growths which are very vascular and develop from the normal synovial fringes. As these develop they fill the entire joint cavity and distend the capsule. The sharp, severe pains are increased by acute inflammatory exacerbations, accompanied by œdema and some redness of the skin and a slight elevation of temperature. Movements become more and more painful and limited as the thickened capsule contracts, as it gradually fuses with the surrounding tissues, and as the margins of the articular cartilages become fibrillated and transformed into fibrous tissue. The joints become considerably swollen. The boundaries of the swelling, which become more pronounced as the muscles atrophy and the contractures develop, are not sharply defined. The swelling often is comparable to that which occurs in tuberculous arthritis (von Volkmann). Hard nodules in the capsule and the hypertrophied villi may be palpated through the soft tissues. When passive movements are made a grating may be felt and heard. If almost all the joints are involved

the patient lies helpless in bed, dying after a number of years of exhaustion.

The third form (arthritis chronica rheumatica ankylo-poetica) is the most advanced. It may be preceded by one of the forms above described or develop independently. The hypertrophied and thickened capsule shrinks and contracts, while the articular cartilages become fibrillated and destroyed by the pressure of the newly formed, vascular masses of connective tissue. The articular surfaces are denuded and become adherent. Bony anchylosis may develop from this fibrous anchylosis, which is accompanied by subluxations and contractures.

Strümpell and P. Marie have described a progressive anchylosis of the spinal column (chronic anchylosing spondylitis) proceeding from below upward which is associated with anchylosis of some of the larger joints. Bechterew has also observed cases of anchylosis of the spine accompanied by pain and symptoms due to compression of the roots of spinal nerves (neuralgias, flaccid paralyses of the muscles of the extremities). The form of anchylosing spondylitis described by Bechterew differs, however, in a good many respects from that described by Strümpell and Marie.

The anatomical investigations of E. Fränkel have shown that both these forms of spondylitis have about the same pathological basis, namely, an inflammation of the small vertebral joints leading to an anchylosis. The periosteal growths are secondary and are due to altered static conditions. Both of these forms, therefore, belong to chronic articular rheumatism and not to arthritis deformans.

Prognosis.—The cure of chronic articular rheumatism is not to be expected. Even the mildest forms may continue through life. The severest forms may, however, be somewhat alleviated except when all the joints are involved and anchylosed.

Diagnosis.—It is often impossible to make an absolute diagnosis between chronic articular rheumatism, chronic gonorrheal arthritis, gout, and arthritis deformans. Often tuberculous arthritis cannot be positively excluded.

Treatment.—The greater part of the treatment of chronic articular rheumatism belongs to internal medicine. Salicylates, hydrotherapy (steam and Turkish baths, hot compresses), massage, and gymnastic exercises have been employed. A prolonged stay at hot springs, such as Teplitz, Wildbad, Gastein, Wiesbaden, Baden-Baden, Hot Springs (Arkansas), White Sulphur Springs (Virginia), and change of residence to a warm, dry climate, are often of value.

Bier's passive hyperæmia lessens the pain and favors the separation of fibrous adhesions. It may be alternated with treatment by the hot-air apparatus (*vide* p. 310). The œdematous infiltration of the tissues and

the improvement of the circulation delay the cicatricial contraction of the tissues and render the joint more movable. According to Büdinger, the injection of sterilized vaselin (1–4 c.c.) into the affected joint is of value.

The contractures may be corrected by gradual reduction by weight and pulley, or by forcible reduction under general anæsthesia. If the joints are painful an attempt should be made to secure anchylosis in a useful position by immobilizing the part in a plaster cast; otherwise, an attempt should be made to prevent anchylosis. Resection should be considered when contractures in poor positions develop. Large capsular growths and hypertrophied synovial fringes should be removed.

(3) ARTHRITIS DEFORMANS—OSTEO-ARTHRITIS CHRONICA DEFORMANS

Nature and Pathology of Arthritis Deformans.—The pathological processes occurring in arthritis deformans differ from those of chronic articular rheumatism, but the clinical pictures at the beginning are often very similar. In arthritis deformans the changes in the cartilages and bones (atrophy and proliferation alternating) are the most prominent, but the capsule and synovial villi also become hypertrophied and thickened as in chronic articular rheumatism. Arthritis deformans also differs from chronic articular rheumatism in the absence of adhesions between the articular surfaces.

In arthritis deformans the articular cartilages become softened, fibrillated, and fissured at the points where they are exposed to the greatest pressure. The bone is then exposed and becomes smooth and polished off by the movements of the joint. Nodular masses of cartilage (ecchondroses), which later become transformed into osteoid tissue and true bone, develop at the margins of the joint. These cartilaginous masses are at first united by a pedicle, but as they enlarge the pedicle becomes thinner, until finally it is destroyed and the cartilaginous masses become free. Floating cartilages may develop in this way. The bone disappears spontaneously by absorption, while the bone-marrow assumes, as the fat is absorbed, a gelatinous, or as liquefaction occurs, a cystic, appearance (Ziegler). The spongy bone lying beneath the articular cartilage becomes softened and yielding, and gradually flattens where exposed to pressure. At the same time bone develops from the periosteum adjacent to the articular cartilage, which unites with the masses developing from the margins of the cartilage to form large, nodular marginal growths. [A characteristic " lipping " of the margin of the cartilage develops in this way.] Capsular changes are associated with these cartilaginous and bony changes. The capsule becomes thickened and shrunken. At times plates and spiculæ of bone develop within it, while the surface of the synovial membrane may be covered with pro-

liferating, hypertrophied villi. The latter may be partly fibrous, partly fatty; often they contain cartilaginous foci. Floating cartilages are often formed by the detachment of thickened calcified villi.

The entire joint becomes greatly changed by these processes. The articular ends of the bones become flattened, broad, and surrounded by an irregular row of osteophytes; the articular surfaces become widened, their margins thickened and irregular. Depressions and cavities alternate with grooved and smooth surfaces. The deformed ends of the bones entering into the formation of the diseased joint are surrounded by nodular masses of bone or cartilage and knoblike tuberosities. Between these masses, in the depressions and grooves, are often found innumerable, larger or smaller, free or pedunculated, bodies.

It may be years before the changes become as marked as described above. The disease is characterized by a slow but steadily progressive course, and although it may remain stationary for a time, a subsidence is extremely rare and has been observed only at the beginning of the disease.

Joints Most Frequently Involved.—Arthritis deformans is most commonly observed in the hip and knee joints, and then in order of frequency in the elbow, wrist, shoulder, and ankle joints, in the spinal column and interphalangeal joints. The disease may develop in a single joint (especially the large ones) or simultaneously in a number of joints. It is more frequent in the male sex. It begins, as a rule, in middle life, but may develop in children and young adults.

Nothing definite is known concerning the cause of arthritis deformans. We only know that injuries to the joints and the factors already mentioned in chronic articular rheumatism play a rôle.

Malum Senile.—This is a chronic disease of the joints occurring in old people and attacking most frequently the hip joint, but also the shoulder and elbow joints. It differs from arthritis deformans in the absence of bony and cartilaginous growths. The atrophy and destruction of the ends of the bones are probably due to senile nutritional disturbances, and resemble the processes occurring in arthritis chronica ulcerosa sicca (Ziegler). The capsule, however, becomes thickened and the synovial villi hypertrophied. Similar changes occur in patients of advanced age whose extremities have been immobilized in dressings for a long time and have not been used.

Symptoms.—Arthritis deformans has an insidious onset. For a long time slight pain, creaking and rubbing of the joint on movement, and a sense of stiffness most marked after keeping the joint at rest for a long time, and disappearing rapidly when the limb is used may be the only symptoms. From time to time a serous exudate is poured out into the joint. The effusion recurs frequently when there are free bodies

in the joint which irritate the synovial membrane. It is frequently the first thing to direct the attention of the patient to the disease. Gradually the form of the joint changes. The joint swells as a result of the thickening of the capsule and enlargement of the bones, irregular prominences forming about it.

The greater the alteration in the shape of the joint, the greater the limitation of motion due to the development of marginal osteophytes which interfere with the normal movements of the bones, the more marked the deformities, such as genu valgum, varum, etc., are. Movements are often very painful when the bony changes are becoming advanced. The joint, in spite of the contraction of the capsule, which may lead to a capsular anchylosis, becomes weak and flail, and finally pathological dislocations and contractures develop.

In spite of the number of disturbances and the steady, progressive nature of the disease, there are no immediate dangers to life.

Diagnosis.—The diagnosis is based upon the thickening of the capsule, the serous effusion, the creaking and rubbing elicited by movements of the joint, the palpable marginal osteophytes and knoblike tuberosities, and upon the presence of free bodies in the joint cavity. In the beginning, arthritis deformans is very similar to chronic articular rheumatism, especially if a number of different joints are involved. Neuropathic joint lesions are usually painless, and a careful examination will reveal the principal disease. Joints attacked by arthritis deformans, which are fixed or but slightly movable, may be easily mistaken for tuberculous joints.

Treatment.—The same methods and drugs are used in the treatment of arthritis deformans and of chronic articular rheumatism. Contractures due to capsular changes, which are infrequent, may be forcibly corrected; if necessary, under general anæsthesia. If the pathological changes in one of the larger joints are advanced and the deformity is marked, resection of the joint may be performed. In resection of the joints of the upper extremity and of the hip an attempt should be made to secure a good range of motion, while after resection of the knee and ankle joints, bony anchylosis in a useful position is desired. Occasionally amputation is indicated in old people, when the destruction of the knee or ankle joint is advanced.

Free Joint Bodies, Floating Cartilages.—Loose bodies in a joint give rise to special symptoms. These free bodies are due to the separation or breaking off of fibrous (later calcified), or cartilaginous synovial fringes, or of ecchondroses. Sometimes they lie in the grooves and depressions in the joints, at other times they glide rapidly hither and thither when movements are made. They are often connected with the articular surfaces or to the synovial membrane by a delicate fibrous pedicle. Free bodies are

frequently found in arthritis deformans, and if the pathological changes are limited in extent the symptoms caused by them may be the most prominent feature. Free bodies may even be found in joints in which no trace of arthritis deformans can be discovered, and where no history of previous injury to the joint can be elicited. König believes that in these cases larger or smaller pieces become separated from the articular ends of the bones, much as a sequestrum is separated, by some process the nature of which is obscure. He has given the name of *osteochondritis diseccans* to this obscure, circumscribed disease of the ends of the bones. The loss of substance in the bone is repaired by the proliferation of the surrounding cartilage. It is still possible that even in these cases a slight trauma preceded the formation of the free bodies.

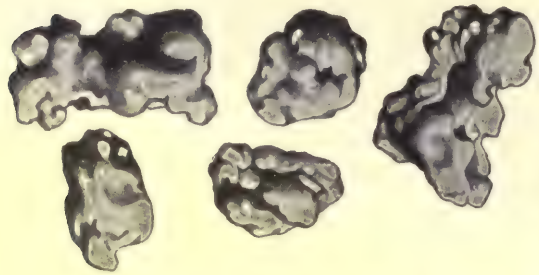


FIG. 271.—FREE BODIES REMOVED FROM A KNEE IN A CASE OF ARTHRITIS DEFORMANS. (Male patient thirty years of age.)

The pathological or arthritic free bodies differ from the traumatic in appearance. The former have a mulberry appearance, which is due to the proliferation of the cartilage surrounding the bony nucleus, while the latter are smooth. They contain hyaline and fibrous cartilage, calcified fibrous tissue and bone. Bone frequently forms the nucleus of the free bodies. If the structure indicates that the body was derived from a normal articular surface, it is probably of traumatic origin. They may occur singly or in large numbers, and vary in size from a pea to a walnut.

Symptoms.—The principal symptom caused by free bodies in the joint is sudden, severe, often agonizing, pain experienced when some particular movement is made. The joint then becomes locked and the patient falls to the ground as a result of the interference with the function of the joint, or faints because of the severe pain. These symptoms, which are not infrequently followed by a serous synovitis, are due to the incarceration of the free body between the articular surfaces.

Joints in which Free Bodies Occur Most Commonly and Diagnosis.—Free bodies are found most frequently in the knee, elbow, and hip joints, especially in powerful men of middle age. The diagnosis is not difficult when the symptoms are pronounced, and a history of a number of previous attacks of locking of the joint can be elicited. If the free bodies are in an inaccessible part of the joint and cannot therefore

be palpated, they may be demonstrated in X-ray pictures unless they consist merely of fibrous tissue or cartilage. [It should be remembered in interpreting X-ray pictures of the knee joint, that there is frequently a sesamoid bone in the internal head of the gastrocnemius which casts a shadow. The shadow cast by this sesamoid bone should not be interpreted as due to a free body.]

Indications for Treatment.—If the free bodies cause symptoms they should be removed. As a rule, these bodies can be removed through a small incision in the capsule. When they can be palpated, cocain anæsthesia is sufficient. Of course these operations should be performed under artificial ischæmia and under the greatest possible precautions to secure asepsis. If there are a number of free bodies a large incision may be required. If during the operation the free body becomes lost in the joint cavity, pressure should be exerted at different points and movements made. Often the free body can be forced out of the incision by proper manipulations. After suture of the incisions in the capsule and in the skin, the joint should be immobilized for a week.

Fig. 272.—Arthritis Neuropathica (Tabica) of the Right Knee and Ankle Joints.

Neuropathic Arthritis.—The joint changes occurring in locomotor ataxia and syringomyelia, occasionally also after compression and injuries of the spinal cord and after inflammation and division of the peripheral nerves, are classified as neuropathic (Charcot joints) and are closely allied to those found in arthritis deformans. In neuropathic arthritis, however, the destruction is much more extensive, the atrophy and proliferation more pronounced, and the course much more rapid.

The *symptoms* frequently begin acutely after exertion or slight trauma with a serous exudate into the joint and an extensive, tense œdema of the para-articular tissues. Neuropathic arthritis differs from all other lesions of the joints in that it is *absolutely painless.* According to Charcot, in a mild or benign case there are but slight changes in the cartilage and bone. In the severe or malignant cases all the structures entering into the formation of the joint are involved. An atrophic and a hypertrophic form may be differentiated.

In the former the ends of the bones become small and atrophic, while in the latter proliferation of the cartilage and bone leads to the formation of marginal osteophytes, of knoblike tuberosities, and of plates of bone within the capsule and surrounding tissues. These two forms may be combined, and as a rule they are, in the same case. In these cases the joints involved become flail as a result of the rapid destruction, and are capable of assuming extreme abnormal positions. Irregularities in form and marked enlargement and expansion of the ends of the bones give to neuropathic joints a very characteristic appearance, upon which alone the diagnosis can often be made (Fig. 272). A serous exudate, free bodies in the joint, spontaneous fractures of the fragile bones, and pathological dislocations complete the picture. The painless development and extent of the pathological changes, the extracapsular formation of bone, and the symptoms of the primary disease enable one to easily differentiate between neuropathic arthritis and arthritis deformans.

Fig. 273.—Roentgen-Ray Picture of Case Represented in Preceding Figure.

Neuropathic arthritis occurring in locomotor ataxia is most common in the knee and hip joints, more rare in the joints of the upper extremity. In syringomyelia, on the other hand, the joints of the upper extremities are most frequently involved, as the lesions

are, as a rule, in the upper part of the spinal cord. Usually but one joint is involved, although the same joint on the opposite side may be attacked.

Trophic disturbances, anæsthesia, and analgesia of the bones and joints, fragility of the bones, and mechanical insults sustained in the ataxic, stamping gait of tabetic patients are important etiological factors in the joint lesions developing in this disease.

The *treatment* is similar to that employed in arthritis deformans. The results following resection, especially resection of the bones of the lower extremity, are not as good as the results obtained

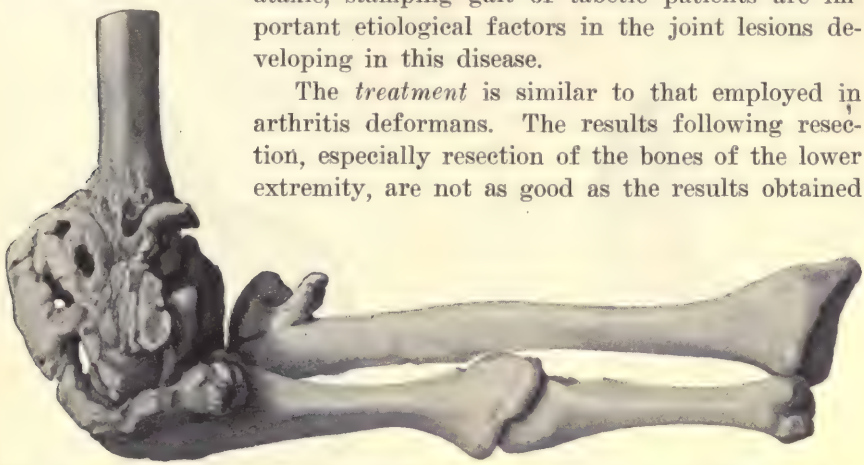


Fig. 274. — Pathological Changes in Elbow Joint in a Case of Syringomyelia. Healed Fracture of the Ulna.

in arthritis deformans, because the bones are atrophic. Better results follow the use of a well-fitting, mechanical support. If the destruction of the joint is very extensive, amputation is to be recommended.

(4) ARTHRITIS URICA, ARTICULAR GOUT

Acute and chronic inflammation of joints form the most prominent features in the clinical picture of gout. It is a constitutional disease, for the most part dependent upon an inherited tendency, in which the metabolic processes are altered and urates are precipitated from the blood and deposited in the tissues, especially in the joints and surrounding structures, causing attacks of inflammation. Chronic alcoholism, high living, and chronic lead poisoning favor the development of gout, which is most frequent in men of middle and advanced age.

Symptoms.—Inflammation of the joints is, as a rule, the first and most important symptom of gout, but changes, of which chronic interstitial nephritis is the most important, may develop in the viscera. Bladder and kidney stones (urates) are frequent, while obesity, arteriosclerosis, and diabetes mellitus not infrequently develop later in the course of the disease.

If the viscera are not diseased, a patient suffering with gout may

attain a ripe old age; on the other hand, chronic nephritis, with its complications, may soon prove fatal.

Gout in most cases has an acute onset, becoming chronic later; rarely is it chronic from the beginning.

Acute Gout.—Acute articular gout usually begins suddenly with severe agonizing pain, increased by movement and pressure, in one joint, as a rule, and with some fever. Sometimes the attack is preceded by

FIG. 275.—ARTHRITIS URICA (GOUT) INVOLVING THE INTERPHALANGEAL JOINTS OF THE LITTLE FINGER. There are masses of urates in destroyed joints, in the tendons, and beneath the skin.

pain in the muscles, weariness, chilly sensations, and indigestion. Gout most frequently attacks the joints of the toes (podagra), preferably the metatarso-phalangeal joint of the great toe. Circulatory disturbances, which are frequent in the terminal parts of the body, the exposed position of the toes, and the frequent occurrence of arthritis deformans in these joints probably predispose them to attacks of gout. The tissues around the joint become swollen, red, shiny, and œdematous, resembling clinically acute suppurative or gonorrheal arthritis. The attacks frequently begin in the middle of the night, but toward morning the pain and fever subside, and the patient sweats profusely. The general condition of the patient remains good. After one or two weeks the night attacks gradually become less and less severe, and the swelling subsides completely, without leaving any noticeable change in the joint.

When the joints have been frequently attacked, covering periods

of months and years, the articular cartilage may become infiltrated with urates; the ends of the bones, ligaments, and peri-articular tissues may become markedly thickened. While these changes are taking place a number of smaller joints may be attacked.

Chronic Gout.—*Pathology.*—Gout may become chronic after the acute attacks have been frequently repeated. In rare cases chronic gout develops without an acute stage, and is accompanied by mild inflammatory exacerbations. Wherever the urates are deposited, the cartilage, and later the bone, synovial membrane, and ligaments may become necrotic. The surrounding healthy tissue proliferates, forming a granulation tissue which surrounds the necrotic tissue and urates, removing or encapsulating them. The thickening of the capsule and anchylosis of the joint are due to the granulation tissue. Abnormal position of the digits and dislocations follow the progressive destruction of the articular cartilages and adjacent bone. The thickening of the joint becomes more marked as the deposit of urates increases.

When the capsule of the joint is destroyed, the urates may extend to structures beneath the skin or form encapsulated masses in the tendon sheaths, bursæ, and subcutaneous tissues. These subcutaneous collections of urates give a peculiar, humped appearance to the joint involved, which justifies the name gout nodules or "tophi" which have been given them. The tense skin covering these tophi may become necrotic or ruptured by injury; then fistulæ form from which chalky masses of urates are discharged, or they may become infected, and suppurative or putrefactive arthritis then develops.

Tophi.—The more or less painful gout nodules or tophi are of diagnostic importance. These are round and covered by a tense, thin skin; they gradually increase in size until they may become as large as a walnut or hen's egg. They are hard and movable upon or firmly adherent to the underlying tissues in which they are deposited. Tophi occur most frequently about the joints of the hands and feet, where they reach considerable size; in the subcutaneous tissue of the scalp, where they can be differentiated from gummatous periosteal nodes only by the fact that they are freely movable; finally, they occur as small nodules, never becoming larger than a pea in the ears, eyelids, and nasal cartilages. The white contents of the tophi shine through the thin skin covering them. If there are no tophi the diagnosis of gout may be difficult, especially in the first few acute attacks, or in cases in which the disease is chronic from the beginning, for gonorrheal arthritis resembles very closely acute gout; chronic articular rheumatism resembles chronic gout. The absence of lymphatic involvement speaks against pyogenic infections.

Fistulæ and marked swelling of the joint suggest tuberculosis. The

chalky, milky character of the secretion and the finding of numerous fine, uric acid crystals give a clew to the correct diagnosis.

The pathological changes in the joints are very characteristic. The articular cartilages look at first, even when there is but little involvement, as if they had been sprinkled over with plaster of Paris or chalk. In time, however, the white substance which is deposited in the groundwork of the cartilage penetrates deeper and deeper, causing a destruction which in advanced cases may even extend to the bone.

The joint then becomes filled with crumbling, mortarlike masses which infiltrate the synovial membrane and capsular ligaments. Small masses of this substance are also found in the peri-articular tissues. They are composed mostly of sodium biurate.

Theories Concerning Cause of Gout.—Nothing definite is known concerning the cause of the precipitation and deposition of sodium biurate in the tissues and the way in which it occurs.

[Futcher, in Osler's "Modern Medicine," speaks of the theories of gout as follows: "Garrod held that in acute gout the alkalinity of the blood is lessened and the uric acid of the blood is increased, owing to the deficient power of elimination on the part of the kidney. The latter is due usually to organic disease, but may be the result of purely functional disturbance. He attributes the deposition of sodium biurate in the tissue to diminished alkalinity of the plasma, which is unable to hold the uric acid combination in solution. During an acute paroxysm there is an accumulation of the urates in the blood, and the local inflammation is caused by their sudden deposition in crystalline form about the joints.

"This theory has had many supporters, and in large part can be accepted, but, as we have already seen, any explanation based on the degree of alkalinity of the blood must be received with some skepticism.

"Sir William Roberts believed that uric acid normally circulates in the form of a soluble quadriurate, which may be represented by the formula $NaHC_5H_2N_4O_3,H_2C_5H_2N_4O_3$, which is sodium quadriurate. The sodium atom may have its place taken by an atom of any of the univalent metals. In the gouty state, according to Roberts, either from deficient action of the kidneys or from overproduction of urates, the quadriurate accumulates in the blood. The detained quadriurate being very unstable and circulating in a medium rich in sodium carbonate takes up an additional atom of the base, and is converted into the biurate as follows: $2(NaHC_5H_2N_4O_3,H_2C_5H_2N_4O_3) + Na_2CO_4 = 4NaHC_5H_2N_4O_3 + CO_2 + H_2O$. The biurate is very insoluble and less easily excreted by the kidneys. It consequently accumulates in the blood, and exists first in a gelatinous and later in the almost insoluble crystalline form. It is then that precipitation is imminent or actually takes place. This

is apt to occur where the circulation is poor and the temperature low and regions in which the lymph contains a relatively high percentage of sodium chlorid, as in the synovial sheaths. This theory has met with opposition from various quarters, and particularly on the part of Tunnicliffe and Rosenheim.

"Ebstein holds that the local manifestations of gout are due to nutritive disturbances which lead to necrosis. He found, after a study of many of the affected tissues in gout, that one change is common to them all, independent of the urate crystallization, and that is a necrosis of the parts in which such deposition takes place. He believes that this necrosis is primary and that it is as characteristic as the biurate deposit. Both changes must coexist in any tissue in order to constitute a true gouty lesion, and he has found such lesions in the kidneys, in hyaline and fibro-cartilage, and in tendons and connective tissue. He calls attention to the early stages of the necrotic process, in which he finds no deposition of the biurates, and consequently maintains that a nutritive tissue disturbance is the primary factor, and uratic deposition a secondary one, in the gouty process, the latter not occurring until death of the tissues takes place. Von Noorden supports Ebstein's views, and believes that the tissue necrosis is due to the action of a special ferment. In recent years attention has been attracted to the xanthin or purin bases as a possible cause of gout. Kolisch found that although the uric acid excretion is diminished, yet the total output of the alloxuric or purin bodies was increased. He believed that the xanthin bases normally are finally oxidized into uric acid in the kidneys, but that in gout the kidneys are diseased and their power to oxidize the xanthin bases is consequently impaired. His results were obtained by methods shown later to be inaccurate, and Sülzer, Laquer, and Magnus-Levy failed to confirm them. Whatever part the xanthin bases may subsequently be shown to play in the etiology of gout, up to the present time they have not been shown to exert an important influence. Undoubtedly some of the xanthin bases are definitely toxic. Kolisch and Croftan have produced arterial and renal lesions by injecting hypoxanthin into animals. Walker Hall confirmed these results and also produced parenchymatous changes in the liver by long-continued injections of hypoxanthin."]

A chronic proliferation of the connective tissues, which encapsulates the necrotic tissue and urates, follows the irritation resulting from the necrosis of the tissues.

Treatment.—The greater part of the treatment of gout belongs to internal medicine. An attempt should be made to prevent the excessive formation of uric acid by regulating the diet, by exercising, bathing, and drinking of alkaline waters.

In acute arthritis the involved joint should be wrapped in cotton or sheet wadding, immobilized, and elevated. Morphin may be required to control the pain; frequently colchicum, sodium salicylate, and other drugs are of value. Moist compresses have a favorable influence upon the pain. They should not be used, however, when there are subcutaneous nodes, as they macerate the skin and may lead to the formation of fistulæ.

The only operative procedures which should be considered are excision of large, troublesome tophi situated in the soft tissues and amputation of deformed and maimed fingers and toes, especially if there is suppuration. Curetting out of the masses of urates is not very often successful, as the tissues are rarely able to form good healthy granulation tissue and to heal, and new masses may be deposited very soon. Not infrequently a new mass is deposited with acute symptoms after an old one has been curetted away. Riedel has, however, observed a permanent recovery in two cases in which the tophi and the capsular ligament were removed from the metatarso-phalangeal joint of the great toe.

(d) DISEASES OF THE JOINTS OCCURRING IN BLEEDERS

Characteristic lesions of the joints are frequent in hæmophilia, a disease in which there is a tendency to hæmorrhages within the joints. These lesions may occur even in childhood, and develop in one or many joints after an insignificant trauma or some physical effort. The knee joint is involved most frequently. If the patient does not die from hæmorrhage from some other source, marked, often serious, changes gradually develop in the joint involved.

Pathology.—According to König there are three stages in the pathological changes, the first of which is a hæmarthrosis. Symptoms of fluid within the joint, resembling closely those of inflammatory hydrops, develop rapidly with slight or severe pain, sometimes with elevation of temperature. After some days discoloration of the skin and ecchymoses indicate the nature of the exudate. If the joint is protected the exudate may be rapidly and completely absorbed. If, however, the exudate is large, a number of weeks may be required for complete absorption. There may be no disturbance of function after the exudate is absorbed. Sometimes patients recover from a number of attacks of this character without any limitation of motion. Gocht observed one case in which there had been forty-five distinct attacks, and still the function of the knee joint was good.

Sometimes earlier, sometimes later, the disease passes into the second stage, that of general chronic inflammation or *panarthritis* (König). The swelling of the joint then does not subside completely after an

attack. Thickening of the capsule, crepitation when the joint is moved, severe pain, limitation of motion, and contractures indicate that the pathological changes are extensive and profound. The fibrin coagulates and becomes organized, maintaining a chronic inflammation of the joint and causing pressure necrosis of the articular cartilages, as in tuberculous arthritis. If the joint is opened (post mortem) there will be found a serohæmorrhagic exudate; a thickened and indurated capsule; brownish, hypertrophied synovial villi in the recesses of the joint; and at the margins of the articular cartilages flat masses of coagulum, often of the thickness of the finger, and irregular defects in the cartilages due to pressure of these pieces of coagulum. Clinically it is scarcely possible to distinguish this form of arthritis from the granulating form of tuberculous arthritis or from the transitional form between tuberculous hydrops and the latter.

The third stage, characterized by regressive changes, leads to the development of contractures. The organized masses of fibrin form an organic fibrous union between the eroded and ulcerated articular surfaces, while the chronically inflamed capsule and peri-articular tissues contract. Contractures and anchylosis, not infrequently accompanied by dislocations, develop. The thickening of the tissues about the joint is the more pronounced as there is more or less muscular atrophy. The thickening about the joint is not due, however, as it often appears to be, to enlargement of the ends of the bone, but to the thickening of the capsule. Even in this stage the diagnosis of tuberculous arthritis is often made.

These three stages do not occur in each patient. In many cases the hæmorrhage ceases before there are any marked changes in the joint, while in others the hæmorrhages are repeated again and again until the deformities and destructive lesions characteristic of the third stage develop.

In making a **diagnosis** other symptoms of hæmophilia are naturally important. The family and previous history of the patient should be carefully elicited, as they are of the greatest importance. As a rule, hæmophiliacs are pale children or young adults of the male sex. Besides tuberculous arthritis, hæmarthrosis following the rupture of a myeloid sarcoma into the joint should also be considered in the differential diagnosis. In these cases the X-ray findings are of great value.

The **treatment** is limited to immobilization and compression of the joint involved, to the use of the weight and pulley, or of an extension apparatus (Gocht) to correct the contractures. Forcible reduction and operative procedures should never be employed. The latter are always associated with the dangers of death from hæmorrhage. Aspiration of

the larger exudates with subsequent washing out of the joint with a three per cent solution of carbolic acid (König) may be done without danger. Concerning the local and general treatment with gelatin *vide* p. 679.

LITERATURE.—*Barth.* Die Entstehung und das Wachstum der freien Gelenkörper. Arch. f. klin. Chir., Bd. 56, 1898, p. 507.—*Bennecke.* Beitrag zur Anatomie der Gicht. Arch. f. klin. Chir., Bd. 66, 1902, p. 658.—*Borchard.* Die Knochen- und Gelenkerkrankungen bei der Syringomyelie. Deutsche Zeitschr. f. Chir., Bd. 72, 1904, p. 513.—*Börner.* Klin. u. path.-anat. Beiträge zur Lehre von den Gelenkmäusen. Deutsche Zeitschr. f. Chir., Bd. 70, 1903, p. 363.—*Büdinger.* Die Behandlung der chron. Arthritis mit Vaselininjektionen. Wien. klin. Wochenschr., 1904, No. 17.—*E. Fränkel.* Ueber chron. ankylosierende Wirbelsäulenversteifung. Fortschr. a. d. Geb. d. Röntgenstrahlen, Bd. 7, 1904.—*Gocht.* Ueber Blutergelenke u. ihre Behandlung. Chir.-Kongr. Verhandl., 1899, II, p. 359.—*Graf.* Ueber die Gelenkerkrankungen bei Syringomyelie. Beitr. z. klin. Chir., Bd. 10, 1893, p. 517.—*Heiligenthal.* Die chron. Steifigkeit der Wirbelsäule (Bechterew) u. die chron. ankylosierende Entzündung der Wirbelsäule (Strümpell), Spondylose rhizomélique (P. Marie). Zentralbl. f. d. Grenzgeb., 1900, p. 11.—*Janssen.* Zur Kenntnis d. Arthritis chronica ankylo-poëtica. Mitteil. aus d. Grenzgeb., Bd. 12, 1903.—*König.* Zur Geschichte der Gelenkkörper in den Gelenken. Chir.-Kongr. Verhandl., 1899, II, p. 1;—Die Gelenkerkrankungen bei Blutern mit besonderer Berücksichtigung der Diagnose. v. Volkmanns Samml. klin. Vortr., N. F., 1892, No. 36.—*Kredel.* Die Arthropathien und Spontanfrakturen bei Tabes. v. Volkmanns Samml. klin. Vortr., 1888, No. 309.—*Linser.* Beitrag zur Kasuistik der Blutergelenke. Beitr. z. klin. Chir., Bd. 17, 1896, p. 105.—*Mermingas.* Beitrag zur Kenntnis der Blutergelenke. Arch. f. klin. Chir., Bd. 68, 1902, p. 188.—*Riedel.* Die Entfernung der Urate und der Gelenkkapsel aus dem an Podagra erkrankten Grosszehgelenke. Deutsche med. Wochenschr., 1904, p. 1265.—*Rosenbach.* Zur pathol. Anatomie der Gicht. Virchows Arch., Bd. 179, 1905, p. 359.—*Rotter.* Die Arthropathien bei Tabiden. Arch. f. klin. Chir., Bd. 36, 1887, p. 1.—*Schmieden.* Ein Beitrag zur Lehre von den Gelenkmäusen. Arch. f. klin. Chir., Bd. 62, 1900, p. 542.—*Schuchardt.* Die Krankheiten der Knochen und Gelenke. Deutsche Chir., 1899.—*Schüller.* Chirurg. Mitteil. über die chronisch rheumatischen Gelenkentzündungen. Chir. Kongr.-Verhandl., 1892, II, p. 404.—*Stempel.* Die Hämophilie. Sammelreferat. Zentralbl. f. d. Grenzgeb., 1900, p. 721.—*v. Volkmann.* Die Krankheiten der Bewegungsorgane. v. Pitha-Billroths Handb. der Chir., Bd. 2, 2. Abt., Erlangen, 1872.—*Walkhoff.* Ueber Arthritis deformans. Verhandl. d. deutsch. pathol. Gesellsch., Sept., 1905, p. 229.—*Ziegler.* Subchondrale Veränderung der Knochen bei Arthritis deformans. Virchows Arch., Bd. 70, 1877.

(e) GANGLION

Ganglion is a term given to a localized cystic formation which develops frequently in the tissues of the capsule of joints, occasionally from a tendon-sheath or tendon.

Occurrence.—Joint ganglia are found most frequently upon the dorsal surface of the wrist, in the depression between the tendons of the extensor indicis and the extensor carpi radialis brevis. As a ganglion develops in this situation it pushes the ligamentum carpi dorsale in front of it. Ganglia are less frequent upon the flexor side of the wrist

joint. When they develop here they are usually situated beneath the radial artery, beside the tendon of the flexor carpi radialis. They also occur on the dorsum of the foot, and occasionally about the knee joint.

The thinned, translucent connective tissue of the capsular ligament forms the wall of the sac, which contains a clear, transparent colloid or gelatinous substance. After unsuccessful attempts at cure the sac contracts firm adhesions with the neighboring tendon sheaths. The cyst is attached to its point of origin by a broad base or short pedicle, and is separated from the cavity of the joint by a delicate membrane, the remains of the joint capsule, unless it has already ruptured into the joint. While old cysts are usually unilocular, recent cysts are multilocular and contain upon their inner wall prominent projecting folds. Even in the walls of unilocular cysts small recesses or cavities can be demonstrated microscopically.

FIG. 276.—GANGLION ON THE DORSUM OF THE FOOT.

Etiology.—Ganglia were formerly considered to be due to the constriction of an evaginated portion of the synovial membrane; in other words, they were regarded as synovial herniæ. This explanation was suggested by Gosselin in 1852. More recently Falkson and Riedel, basing their observations upon clinical experience, have shown that the cysts originate within the tissues of the capsular ligament and not within the synovial membrane. Virchow and von Volkmann had previously suggested that this might be the case. The histological investigations of Ledderhose, and later those of Ritschl, Thorn, and Payr, have verified the clinical findings of Falkson and Riedel. They have found that these cysts are the result of degenerative changes in the capsular and paraarticular tissues (more rarely in the tendinous and paratendinous tissues) resulting in the formation of a gelatinous substance, the contents of the ganglion. Nutritional disturbances, caused apparently by an obliterating endarteritis of traumatic origin, precede the degenerative changes. Later several of these small cysts fuse to form one large one.

Ganglia occur most commonly in young people, during and after the age of puberty, but may develop even at an advanced age. They are more frequent in the female than in the male.

Symptoms.—The first symptoms are indefinite for a long time. Sometimes impairment of motion, at other times neuralgic pains direct the attention of the patient to the swelling, which is supposed to be the result of a sprain. As a matter of fact, ganglia are frequently caused by insignificant lacerations or injuries due to overexertion of the wrist in piano playing, rowing, fencing, etc. The cyst slowly enlarges, but rarely becomes larger than a walnut. Frequently it remains of the same size, and often becomes somewhat smaller spontaneously. Not infrequently the smaller ganglia subside completely without any treatment. A slight limitation of motion may be the only symptom of even the larger ganglia.

The appearance of a ganglion is very characteristic. The smooth or uneven surface of the cyst, which is firmly attached to the surrounding structures or is slightly movable when attached by a pedicle, is covered by normal skin. The smaller cysts may be hard and non-fluctuating. Fluctuation can be easily elicited in the larger cysts.

The **diagnosis** can be made upon the position of the swelling and the characteristics above given. It is important to differentiate ganglia from hygromas of the synovial sheaths and bursæ.

Treatment.—In the treatment an attempt should first be made to cure ganglia by non-operative methods. The author has repeatedly cured ganglia by rupturing them by one blow with a wooden hammer, or by binding a lead button over the swelling until the wall of the cyst has become thinned and has ruptured subcutaneously. Recurrences—large cysts developing from small accessory ones—are frequent after this method of treatment. If the treatment is repeated a permanent cure may be obtained.

If the non-operative treatment has been unsuccessful, an operation, which should be performed with the greatest aseptic precautions, should be advised. In the complete removal it may be necessary to open the joint or synovial sheath, and even the mildest infection may be followed by most serious results. The operation should always be performed under artificial ischæmia, as in this way the anatomical relations may be better exposed, and the fingers should not come in contact with the wound.

Recurrence follows extirpation only when a part of the pedicle or some of the diseased tissue of the capsule is left.

Subcutaneous discission with the tenotome, aspiration and subsequent injection with alcohol and carbolic acid, and incision combined with tamponing are sometimes but not uniformly successful.

LITERATURE.—*Franz.* Ueber Ganglien in der Hohlhand. Arch. f. klin. Chir., Bd. 70, 1903, p. 973.—*Küttner.* Zur Klinik der Ganglien. Zentralbl. f. Chir., 1905, p. 1333.—*Ledderhose.* Die Aetiologie der karpalen Ganglien. Deutsche Zeitschr. f.

Chir., Bd. 37, 1893, p 102.—*Payr.* Beiträge zum feineren Bau und der Entstehung der karpalen Ganglien. Deutsche Zeitschr. f. Chir., Bd. 49, 1899, p. 329.—*Ritschl.* Beitrag zur Pathogenese der Ganglien. Beitr. z. klin. Chir., Bd. 14, 1895, p. 557.—*Thorn.* Ueber die Entstehung der Ganglien. Arch. f. klin. Chir., Bd. 52, 1896, p. 593.

CHAPTER VII

DISEASES OF BONE

(a) CONGENITAL DEFECTS IN SKELETAL DEVELOPMENT

THERE are a number of malformations due to the failure of development of bones. These may be due in part to arrested (aplasia), in part to the inhibition of normal development. Examples of such malformations are complete or incomplete absence of bones of the extremities, of the clavicle and sternum, or defects in the skull bones and vertebræ and fissures in the maxilla. The loss of distal parts of the extremities due to constriction by amniotic bands is also classified with the failures in development. Malformations may be due to excessive development. The most common examples are supernumerary phalanges, metacarpal and metatarsal bones, cervical ribs and additional vertebræ (in tail formation).

Atrophy of the bones (the result of intra-uterine lesions or fractures), the different hypoplastic and hypertrophic conditions of bone which are present at birth or develop soon after are frequently the causes of malformations.

Hypoplasia may affect the entire body, in which case a dwarf results, or a portion of it only, giving rise then to imperfect formation of single parts or organs, such as hypoplasia of the extremities (micromelia) and *congenital skeletal atrophy* (so-called fœtal rickets).

Fœtal Chondrodystrophy and Periosteal Dystrophy.—There are two varieties of fœtal rickets. In the one (cartilaginous dysplasia, fœtal chondrodystrophy of Kaufmann, fœtal cretinism of Horsley) there is impaired endochondral bone formation, while the periosteal bone formation is normal. The bones are therefore thick and hard, but shortened and distorted. In the other (periosteal dystrophy) the periosteal bone formation is interfered with, while endochondral bone formation is normal, and the bones are malformed, soft, and fragile. Hypertrophy of bones may be the cause of general (macrosomia) or partial giant growth. The latter may be confined to parts of the feet or hands.

Bone Changes in Cretinism.—The more or less imperfect development of bones in *cretinism,* a disease occurring endemically in young people

living in goiter regions, is due, as are the other symptoms of the disease (myxœdema, hypoplasia of the genitalia), to a disturbance of the function of the thyroid gland, which is either absent, atrophied, or altered in structure (goiter). The relationship between cretinism and the thyroid gland has been demonstrated experimentally (Hofmeister, von Eiselsberg). In cretinism the epiphyseal cartilages do not become ossified for a long time, but they are unable, because of regressive changes, to produce bones of normal length. The centers of ossification in the epiphyses develop late. If the feeding of thyroid gland or thyroid preparations is begun early and continued the symptoms may improve and the growth of the bones may be increased.

LITERATURE.—*v. Eiselsberg.* Die Krankheiten der Schilddrüse. Deutsche Chir., 1901, Kretinismus, p. 197.—*Nasse.* Die Krankheiten der unteren Extremitäten. Deutsche Chir., 1878, Riesenwuchs des Fusses, p. 1.—*Schuchardt.* Die Krankheiten der Knochen und Gelenke. Deutsche Chir., 1899, p. 58.—*P. Vogt.* Die Krankheiten der oberen Extremitäten. Deutsche Chir., 1881.

(b) ATROPHY OF BONE

Atrophy of bone occurs in the form of lacunar resorption. The resorption in diseased bones is not actually in excess of that occurring in normal developing bone (Pommer), but only relatively so, as new bone is not formed to replace that lost by resorption (Schuchardt).

Concentric and Eccentric Atrophy of Bone.—Sometimes the resorption begins upon the surface of the bone and extends inward, while at other times it begins in the medulla and advances outward. In the former (concentric atrophy) the bone becomes thinner and smaller, while in the latter (eccentric atrophy) the medullary cavity, the Haversian canals, and the spaces in the spongy bone become enlarged as the trabeculæ of bone disappear and fat accumulates in these enlarged spaces. If the entire bone becomes porous and light the condition is known as *osteoporosis*; if decayed and fragile, as *osteopsathyrosis*; if, after considerable loss of calcium salts, it becomes flexible, as *osteomalacia.*

The fragility of atrophic bones is of greatest surgical interest, as the diseased bones may be fractured by very slight injuries, even by bearing the body weight upon them.

Delayed Repair After Fracture.—Repair of such fractures is often delayed; non-union is frequent, except in the pathological fractures occurring in neuropathic atrophy, as there is but little tendency to the formation of callus. For the same reason it is often difficult to obtain union after operation upon joints in which anchylosis (e. g., paralyzed extremities) is useful and desired.

Causes of Atrophy of Bone.—There are a number of different causes of atrophy of bone. Some of these may be local, the majority are general.

Among the local causes are aneurysms, tumors, and echinococcus cysts. These develop either without the bone and later extend to it, producing a pressure necrosis of the cortex, or within the bone, and as they enlarge cause a pressure necrosis and expansion of the cortex, finally rupturing through it. The atrophy following long-continued non-use (atrophy of disuse) is also placed in this class. Disuse atrophy is most frequent in the bones of the extremities, which have been thrown out of function for a long time or permanently as the result of severe injuries, inflammatory lesions, paralyses, or after fractures with vicious union in which the extremity cannot be used to support the weight of the body. The conical form which the bones in an amputation stump assume may be prevented if an artificial limb is used and weight is borne upon the stump.

Nutritional and trophic disturbances are the most important of the general causes. In old age a general osteoporosis occurs, and fractures, especially of the neck of the femur, following insignificant injuries and curvature of the spine are frequent. Atrophy of the alveolar borders of the jaws following extraction of the teeth, and shortening of the lower parts of the face are the best-known examples of this form of atrophy of bone. Superficial, sometimes perforating, defects of the bones of the skull may result from senile atrophy. Similar changes occurring in young people suffering from chronic infectious diseases are known as *marantic atrophy* of bone.

If the cause of the atrophy depends upon some disease of the peripheral nerves or central nervous system, it is called *neurotic atrophy*. This form of atrophy is due wholly or in part to the loss of trophic influences. It is indicated in growing bones by shortening, in fully developed bones by osteoporosis, sometimes accompanied by osteomalacia. Of course if there is paralysis, disuse is also an etiological factor. The pure form of neurotic atrophy is found only in diseases, such as syringomyelia, locomotor ataxia, and paretic dementia, in which the use of the extremities is not interfered with. Painless, spontaneous fractures are frequent in this form of atrophy. Often the repair of these fractures is rapid, and excessive callus is formed, as the bones are analgesic and mechanical irritation of the fractured ends is not prevented by pain as in ordinary fractures.

Sudeck has shown by Roentgen-ray pictures that the bony atrophy developing acutely after injuries and inflammation is due to trophic disturbances. The rapid loss of the contour of the affected bone in these cases cannot be explained by disuse alone. It is probably of a

reflex nature, similar to the muscular atrophy occurring in arthritis (*vide* p. 655).

After the primary lesion has healed the atrophy may gradually subside. In tuberculous arthritis it is often difficult to determine whether the indistinct, clear shadows in the X-ray pictures correspond to tuberculous foci or to atrophic bone. If due to atrophy the shadows will be much more extensive.

Idiopathic Osteopsathyrosis.—Idiopathic osteopsathyrosis is a peculiar but rare form of atrophy of bone, the cause of which is unknown. The disease, characterized by frequent, sometimes multiple fractures, develops in early childhood. Schuchardt writes of a girl twelve years of age who had sustained forty-one fractures in ten years, the first one occurring when she was two years old. In a large proportion of cases the disease is inherited.

Inflammatory atrophy of bone, the result of rarefying osteitis, is found in pyogenic, tuberculous, and gummatous lesions of bone (see p. 425).

LITERATURE.—*Adler.* Ueber tabische Knochen- und Gelenkerkrankungen. Sammelreferat mit Lit. Zentralbl. f. d. Grenzgeb., 1903, p. 849.—*Schlesinger.* Die Erkrankungen der Knochen und Gelenke bei Syringomyelie. Zentralbl. f. Grenzgeb., 1901, p. 625.—*Schuchardt.* Die Krankheiten der Knochen und Gelenke. Deutsche Chir., 1899, pp. 58–83.—*Sudeck.* Ueber die akute (reflektorische) Knochenatrophie nach Entzündungen und Verletzungen an den Extremitäten und ihre klinischen Erscheinungen. Fortschritte auf dem Gebiete der Röntgenstrahlen, Bd. 5, 1902, p. 277;—Zur Altersatrophie (einschl. Coxa vara senium) und Inaktivitätsatrophie der Knochen. *Ibid.*, Bd. 3, 1900, p. 201.

(c) HYPERTROPHY OF BONE

Hypertrophy of bone is frequently the result of inflammatory processes, which lead (especially in syphilis) to the formation of hyperostoses, in periosteal tumors and in ulcers adjacent to bone to the formation of osteophytes, in suppurative osteomyelitis to the formation of the involucrum, and in fractures to callus formation. Suppurative and tuberculous lesions of bone occurring during the period of growth often lead to an increase in length of the bone involved.

Leontiasis Ossea.—There are but two forms of independent hypertrophy of bone, and these are rare: Leontiasis ossea and acromegaly. The disease called leontiasis ossea by Virchow begins in young people without any distinct symptoms, and gradually leads after a number of years to a symmetrical thickening and induration of the bones of the face and skull. The changes usually begin in the maxillæ. The skeleton of the face gradually becomes transformed into a heavy, bony mass, and all semblance to normal human features is lost. The symptoms

which follow the painless proliferation and hypertrophy of the bones are secondary, as the skull bones of from 4 to 5 cm. in thickness press upon the brain, causing headaches, convulsions, paralyses, and mental disturbances. The thickening of the facial bones occludes the nasal passages, forces the eyes out of the orbit, causing exophthalmos, and destroys the optic nerves, causing blindness. The foramina and canals through which the cranial nerves pass are narrowed and symptoms of pressure result (loss of sense of smell, trigeminal neuralgia, etc.). Frequently suppurative dacrocystitis and erysipelas of the face and head precede the development of the disease, but it is not probable that they have any etiological relation to it. Treatment has no effect upon the course of the disease. Even resection of the bones first involved does not delay its progress.

FIG. 277.—LEONTIASIS OSSEA.

Acromegaly was first described by P. Marie in 1886. In this disease there is not only a hypertrophy of the bones of the distal parts of the extremities, of the skull and face due to a proliferation of the periosteum, but also a thickening of the soft parts. In the head the hypertrophy affects most commonly the lower jaw, nose, lower lip, and tongue. In some instances the penis or clitoris is hypertrophied. [In a number of cases, the external genitalia have been smaller than normal.] The disease begins in young or middle-aged people and produces changes in the physiognomy and in the shape of the feet and hands which are very characteristic. The disease is supposed to be due to diseases of the hypophysis (tumors, hypertrophy, cysts, and sclerosis). [" Tamburini's suggestion that acromegaly is dependent upon excessive function of the hypophysis is highly important. As yet no cases of undoubted acromegaly have been reported in which changes in the gland were absent upon both gross and microscopic examinations, and in those instances where necrosis and softening (probably post mortem), sclerosis, colloid degeneration, etc., have been found, no mention is made of the relation between chromophile and chromophobe elements. Experimental removal of this gland, its destruction by neoplasm, infectious granulomata, and aneurysm do not produce the disease, so that it seems proper to assume that acromegaly is not dependent upon an abolished or lessened function of the hypophysis."—Lewis, *The Johns Hopkins*

Hospital Bulletin, Vol. XVI, May, 1905, p. 164.] Paræsthesias and slight pain in the extremities, loss of the finer sensations in the hands and feet, and loss or decrease in sexual desire are common. The hands and feet gradually become clumsy and pawlike or spadelike, as the bones become thickened by the formation of new bone, which is most pronounced near the ends. The bones of the forearms and legs also become considerably thickened. The changes in the face are most striking, as the lower jaw enlarges and projects beyond the upper, as the lips, eyelids, nose, ears, tongue, and cheeks become thickened, often causing folds in the skin. Gradually kyphosis develops, while the bones of the trunk widen. Cachexia gradually develops, the heart and large vessels become diseased, and the disease terminates fatally after a number of years. Acromegaly is especially frequent in giants. The only relation between gigantism and acromegaly is that the former seems to predispose to the development of the latter. According to Arnold there is no increase in the length of bones in acromegaly.

Acromegaly cannot be mistaken for leontiasis ossea if a careful examination is made, as in the latter the fingers and toes are not involved. Usually arthritis deformans can be easily differentiated, as the changes in acromegaly, although they involve the ends of the bones, are extra-articular (*vide* Schuchardt).

Similar changes, involving especially the terminal phalanges of the fingers and toes, and the epiphyses of long, hollow bones, occur in young children suffering from chronic diseases of the heart and lungs (Bamberger). The enlargement is due to the proliferation of the periosteum. P. Marie has grouped these pathological manifestations and described a distinct clinical entity which he calls *osteoarthropathie hypertrophiante pneumique.*

LITERATURE.—*Bardenheuer* und *Lossen.* Leontiasis ossea Kölner Festschrift, 1904, p. 154.—*Mauclaire.* Maladies non traumatiques des os. Traité de chir., le Dentu et Delbet. Paris, 1896. T. II, p. 723.—*Schuchardt.* Die Krankheiten der Knochen und Gelenke. Deutsche Chir., 1899, pp. 150, 225.

(d) RICKETS

Definition.—Rickets from the Greek ῥάχις, meaning spine) is a general disease of malnutrition occurring in children and manifesting itself mainly in lesions connected with the bones. It usually commences within the first three years of life, but sometimes appears later (Rose and Carless, "Manual of Surgery," p. 597). The disease was first accurately described by Glisson, an Englishman, and therefore the disease is often referred to in Germany as the English disease.

Pathological Changes.—Rickets is characterized by changes in normal bone development consisting of an excessive formation of osteoid tissue which is prepared for bone formation, a diminished deposit of lime salts in this tissue, and an increased resorption of newly formed bone. The most striking symptoms of the disease are *enlargements* of the *epiphyses* due to broadening of the epiphyseal cartilages, and the *development* of *deformities*, the result of *softening* and *flexibility* of the bones. The calcium content of the bones is reduced more than one half.

Osteoid tissue develops upon the surface of the bone and in the medulla, especially upon the metaphyseal side of the epiphyseal cartilages, forming vascular, spongy, soft, grayish red masses of tissue. The periosteal deposits of this osteoid tissue are localized and may be removed with the thickened periosteum. The myelogenous deposits in severe cases are not localized, but are distributed throughout the entire metaphysis. Normal endochondral bone formation is greatly altered. The epiphyseal cartilages are greatly widened, and there is a considerable increase in the number of columns of proliferating cartilage cells. Normally the epiphyseal cartilage is a well-defined, bluish white or white line, cartilage and bone being sharply differentiated from each other. In rickets the epiphyseal cartilage becomes broadened and irregular, its sharp outlines are lost and medullary spaces and osteoid tissue extend into the cartilage, and the delicate white streak indicative of primary calcification disappears. The epiphyseal cartilages appear broad, and are provided with irregular processes and outgrowths which may become separated to form islands of cartilage within the osteoid tissue. Bony trabeculæ and calcified cartilage are also found within the latter. [The development of multiple osteomata or chondromata is supposed to be secondary to rickets, the displaced island of cartilage forming the nuclei for these benign tumors.]

There is also an increased lacunar resorption of the newly formed bone, and in this way the medullary spaces and the Haversian canals become enlarged, an osteoporosis developing. Areas may be found in flat bones in which the normal bone is entirely replaced by osteoid tissue. Decalcification may be as marked as in osteomalacia (von Recklinghausen).

Calcification of this osteoid tissue does not occur at all or intermittently, depending altogether upon whether the disease progresses without abatement or improves. As the patient is recovering from the disease, the osteoid tissue becomes transformed into hard sclerotic bone, which may completely occlude the medullary cavity. The deformities which may have developed then become permanent. Slight bending of the bones may be corrected during subsequent growth.

Etiology.—The cause of these alterations in the development of bone is unknown, notwithstanding the number of investigations that have been made. One view held by Pommer, Heubner, and Zweifel is that the deficient calcification of the osteoid tissue is the result of nutritional disturbances. Kassowitz ascribes the changes to a chronic inflammatory hyperæmia, the cause of which is unknown. A number of important objections have been raised against each of these views. In spite of this, one cannot help thinking that the changes are due to the action of some toxic material which accumulates in the blood, as the result of the loss of function of some one of the ductless glands (according to Stöltzner, possibly the suprarenal) and acts upon the bone, especially upon the articular ends where there is a physiological hyperæmia. It can be definitely stated, in spite of the fact that the breast-fed children of the well-to-do classes are not spared by the disease, that insufficient or improper food is an important etiological factor, and that poor hygienic conditions, the want of air and light, uncleanliness, and intestinal catarrh predispose to it.

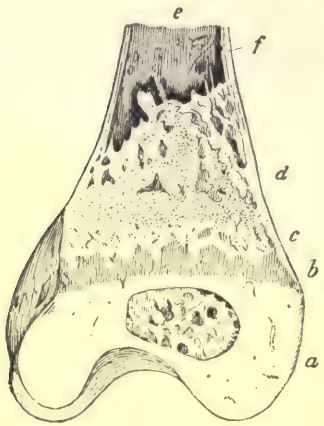


FIG. 278. — CORONAL SECTION THROUGH THE LOWER END OF THE FEMUR OF A CHILD TWO AND A HALF YEARS OF AGE SUFFERING WITH RICKETS. (After Kaufmann.) *a*, Lower epiphysis, normal cartilage; *b*, mottled, bluish red, swollen, soft zone of proliferating cartilage; *c*, zone in which the vessels and medullary spaces with osteoid tissue have penetrated the soft cartilage; *d*, osteoid tissue; *e*, dilated medullary cavity with but little spongy bone; *f*, thinned compact bone covered by a layer of osteoid tissue.

The disease develops most commonly during the second year of life; very rarely after the fifth or sixth. The cases observed during the fifth and sixth years are usually merely exacerbations of mild cases which have persisted for some time. It is a disputed question whether the changes observed in rickets are ever congenital. The changes observed in the so-called fœtal rickets (p. 733) have no relation whatever to the changes found in the disease under consideration. In the so-called late rickets (rachitis tarda) developing at puberty, the bones become soft and yielding, and deformities such as curvature of the spine (habitual scoliosis) and of the ends of the long bones (genu valgum and varum adolescentium, coxa vara) develop, the softened bones yielding under the weight of the superimposed parts. The pathological changes in late rickets differ from those occurring in earlier life, being limited to that part of the metaphysis, poor in lime salts, immediately adjacent to the hypertrophied epiphyseal cartilage (Fig. 279).

The severest and most resistant cases occur chiefly in the children of the poor classes living in cities, among whom the disease is also most common. The disease is rare among the children of the middle classes, and if it does develop it is mild and is easily cured.

Onset and Changes in Bones.—It is not possible to state definitely when the disease begins, as it has an insidious onset. *Frequent* and *profuse sweating, tenderness* of the *bones, anæmia,* and *myasthenia* are recognized as prodromata by physicians experienced in children's diseases. Suspicion may be aroused when the child first attempts to walk.

The course of the disease is always chronic. As a rule the earlier the disease develops the more rapid the course. In the beginning the rapid involvement of the different bones is often quite striking. From time to time the symptoms subside, but exacerbations are frequent, especially during the winter months, when the hygienic conditions are apt to be poor.

The osteal symptoms vary a great deal, and only in the severest cases are they equally prominent in all the bones. In these cases growth is retarded (rachitic dwarf), the epiphyses become enlarged and expanded, the flat bones become thickened, sometimes atrophied, and deformities of the long, hollow bones, caused by muscular action or by the weight of the superimposed parts, develop and fractures may occur. In the milder cases growth is scarcely interfered with, there is less tendency to bending of the bones, deformities are wanting, or if they do develop they are limited to the ends of the bones (e. g., genu valgum), and the enlargement of the epiphyses is not marked.

Fig. 279.—Genu Valgum Adolescentium. (From a patient seventeen years of age.)

Changes in the Skull Bones.—In the skull, especially in the occipital regions, the bones may become soft and yielding, and as a result of the loss of bone, some portions may become membranous again (cranio-tabes). The fontanelles are wide and may remain open for a long time, until the third or fourth year. The head is large, and the forehead appears square in shape, while the parietal and frontal eminences are enlarged by deposits of osteoid tissue beneath the periosteum.

Sometimes the changes in the skull are associated with hydrocephalus. The hard palate becomes high and arched, the alveolar border of the maxilla projecting forward like a beak, while the symphysis of the mandible becomes flattened. The teeth do not erupt until late, are stunted, defective in enamel, and decay early.

Changes in the Thorax.—In the thorax there develops at the junction of the ribs with the cartilages a row of round nodules (rachitic rosary) which may often be seen through the skin. If there is any obstruction to the entrance of the air (tracheitis or bronchitis) the atmospheric pressure may cause the softened bones and cartilages to sink in, and as a result the sternum is pushed forward, producing a typical deformity known as the " chicken breast " or pectus carinatum. The natural curves in the clavicle may also be accentuated. A transverse groove (Harrison's), corresponding in position to the attachment of the diaphragm, often develops across the lower part of the chest. It is produced by the traction of this muscle upon the softened ribs and cartilages. The projection or flaring of the ribs below this line is caused by the enlargement of the abdominal viscera.

A kyphosis develops in the lower dorsal and lumbar regions, especially in children who are carried a great deal. [This kyphosis, which extends over a number of vertebræ, is never angular as in tuberculosis.] Scoliosis is rare.

Changes in the Pelvis.—The changes in the pelvis may be marked, but are of interest chiefly to the obstetrician, as they may interfere with childbirth. The pelvis becomes flattened from before backward, and the cavity becomes contracted as the promontory of the sacrum projects forward and downward and the bone surrounding the acetabulum is forced inward and the symphysis forward.

Changes in the Bones of the Extremities.—The changes in the bones of the extremities are the most striking. Tender thickenings may be palpated upon the ends of the long, hollow bones (especially upon the carpal ends of the radius and ulna and upon the malleoli) which are not covered by thick soft tissues. The entire or part of the diaphysis becomes bowed. The bowing is most common in the femur and tibia, being most commonly forward and outward. The lower third of the tibia bows forward, and may become so flattened from side to side that it resembles a saber sheath. The angular deformities developing at the metaphysis or in the diaphysis are secondary to green-stick fractures. If the direction of the articular ends of the bones is changed by a bending of the softened metaphysis, typical deformities, such as genu valgum, varum, recurvatum, rachitic flat-foot, and coxa vara, develop, the diaphysis remaining normal or becoming bowed. [The bowing of the diaphysis in these cases is usually secondary and compensatory.]

Similar deformities in the bones of the upper extremity occur only in children who have been accustomed to creep about on all fours.

Sclerosis of Bones When Disease Improves. — When the acute stages of the disease have passed and improvement begins, sclerosis of the bones occurs. This process of hardening may be frequently interrupted by exacerbations of the disease. As the sclerosis progresses the epiphyseal enlargements become smaller, the fontanelles close, growth becomes more rapid, and the general condition improves.

Spontaneous Correction of Deformities.—The deformities gradually improve, rarely remaining as great as they were at the time they developed. It has been observed for a number of years that the improvement is gradual, and that, as a rule, the deformities have become less marked or have completely disappeared at the age of puberty. The more rapid the growth, the more rapid the improvement in the deformities is. Usually from two to four years are required for the correction. Clinical observations made by Schlange and Veit in von Bergmann's clinic have shown that the deformities in rachitic children whose growth has not been stunted or retarded subside spontaneously and completely in the sixth or seventh year. According to Kamp's investigations, this occurs in seventy-five per cent of the cases. In cases in which the growth has been stunted, some improvement may occur, but considerable deformity remains. After the age limit above mentioned has been reached, an increase in the de-

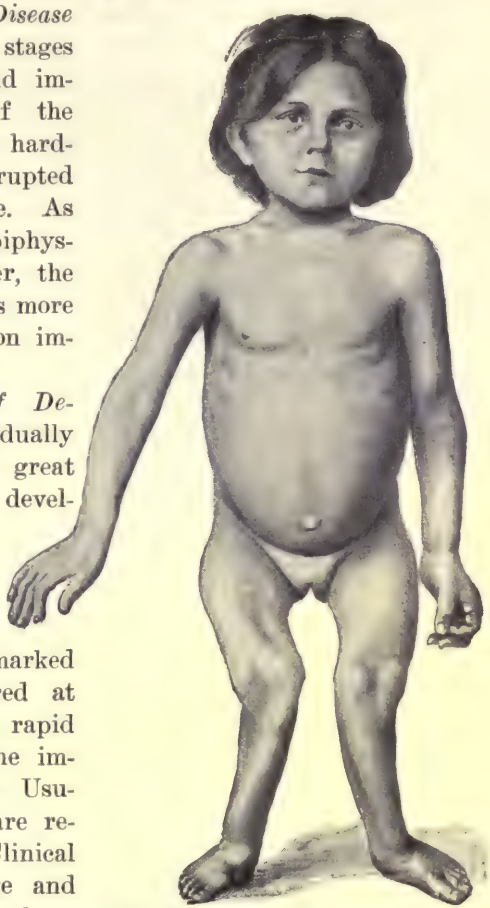


FIG. 280.—RICKETS. The changes are most pronounced in the legs which are considerably shortened. The thighs are rotated outward because of the distortion of the neck of the femur. The shaft of each femur is bent forward and outward. The tibiæ at the position of the upper metaphyses are bent inward, producing genua valga. The deformities at the lower metaphyses, resulting from a bending forward, are less pronounced. Other changes associated with rickets, such as double flat-foot, some thickening of the lower epiphyses of the radius and ulna, widening of the costal arch, and the rickety rosary, are also present.

formities cannot be determined, even when the disease persists, but the bones remain shortened, are capable of but little growth, and the patient remains a dwarf. Some bowing of the diaphysis and enlargement of the epiphyseal ends of the bones may remain permanently in even the most favorable cases, and often are indicative of a previous rickets.

Changes in the Bones Shown by X-Rays.—Recently X-ray pictures of the diseased long bones have shown a number of other changes besides the deformities. The epiphyseal cartilages are broad, irregular, and fibrillated, the cortical layer of bone is thin, and in it are indistinct, localized shadows due to the absence of lime salts. If healing has occurred, the epiphyseal lines become almost as narrow as in healthy bone, and delicate, parallel streaks (calcification lamellæ) running out from the epiphyseal cartilage appear in the metaphysis. A thickening is found in the cortex which is most marked upon the concave side of the deformity, as the greatest weight is placed upon this side.

General Symptoms.—The general symptoms may be mild or severe, and vary from a slight muscular weakness and atrophy to a decided anæmia and emaciation. Intestinal disturbances (meteorism with diarrhœa or obstipation), a tendency to catarrhal inflammations of the lungs resulting from narrowing of the thorax, swelling of the lymph nodes, sweating, eczema, and finally, nervous disturbances, such as unrest, convulsions, and laryngeal spasm, are frequent.

The principal dangers which accompany the severer forms of rickets are weakness and the loss of resistance to infections. Catarrhal inflammations of the lungs, diarrhœa, and infectious diseases may run rapid and fatal courses. Complications such as tuberculosis and syphilis are to be especially feared. Many rachitic children die of some of the acute infectious diseases of childhood. The disease tends to undergo spontaneous cure, and even the deformities which have developed in earlier times may be corrected or improved as the patient grows. Some of these deformities remain, however, and may give rise to serious complications in later life (e. g., rachitic pelvis in childbirth).

Diagnosis.—The diagnosis is usually not difficult, even when the symptoms are not pronounced. The X-ray findings, when positive, are so characteristic as to leave no doubt as to the diagnosis.

Treatment: Medicinal and Surgical.—The treatment in the beginning belongs to internal medicine. In the treatment, as well as in the prophylaxis, special emphasis should be laid upon the improvement of the hygienic conditions and the nutrition of the patient. Phosphorus is supposed by many to have a favorable influence upon the diseases. Kassowitz, basing his conclusions upon animal experiments, believes

that phosphorus, which was advised by Wegner in the treatment of this disease, has no action at all.

Surgery has to do with the reduction and dressing of fractures which occur during the disease and with the correction of deformities. The

FIG. 281.—X-RAY PICTURE OF DEFORMED, RACHITIC BONES OF THE LEG (HEALING HAS OCCURRED). The epiphyseal cartilages are almost normal. The cortex is especially thickened upon the concave side of the tibia.

deformities may be corrected (at one or more sittings) by osteoclasis, using manual force or special osteoclasts, or by linear osteotomy if the deformity is not great, by cuneiform osteotomy if it is. These procedures are usually employed after the sixth year of life, before this time only when the deformities are excessive or when the sclerosis develops early and is marked. If the bones are hard and sclerotic, osteotomy as a rule is to be preferred to osteoclasis. In recent cases any of these procedures are to be avoided as the subsequent immobilization in

plaster-of-Paris dressings, which is required, increases the muscular atrophy, and besides, most of the deformities undergo spontaneous correction. Orthopedic apparatus, which by traction or pressure prevents bowing of the bones, is to be dispensed with for the same reasons. Curvature of the spine and rachitic flat-foot demand early support (plaster-of-Paris jackets and flat-foot arches), as they usually are permanent.

The child should be allowed neither to run about nor be carried as long as the bones remain soft. As the bones become hard the child should be allowed to play, when possible, in the open air. In this way the development of muscles is favored, growth becomes more rapid, and the general condition improves.

Barlow's Disease.—A disease known as *infantile scurvy* (Barlow's or Möller's disease and hæmorrhagic rickets), which involves bone, is sometimes associated with rickets and at other times occurs as an independent affection. It is regarded by some as a hæmorrhagic form of rickets, by others as a peculiar form of scurvy. Nothing is known concerning its cause and exact nature.

It begins with severe pain in the bones, which is soon followed by swelling due to subperiosteal hæmorrhages. These hæmorrhages are most common in the lower end of the femur and in the upper end of the tibia; rare in the bones of the upper extremities and the bones of the trunk. Exophthalmos may develop, being caused by retrobulbar hæmatomas. Hæmorrhages from the gums and skin and hæmaturia complete the picture of the hæmorrhagic diathesis. Osteoporosis follows the imperfect ossification and progressive resorption of bone, and pathological fractures and separation of the epiphyses are frequent. Hæmorrhages may occur in the altered, gelatinous, and fibrous bone marrow poor in cells.

The disease is most common in children from one half to one year of age who have been poorly nourished or have been fed with poor artificial foods. Severe anæmia and great weakness develop rapidly, and the disease ends fatally after some weeks if the food is not changed and properly modified cow's milk given. With proper diet the general condition gradually improves until recovery is complete.

There is little of surgical interest in this disease except the treatment of the fractures which occur, and making the diagnosis between it and inflammatory separation of the epiphysis and bone tumors. The development of multiple osteal swellings, of hæmorrhages into the skin and from the mucous membranes enables one to make a positive diagnosis.

LITERATURE.—*Hauser.* Grundriss der Kinderheilkunde. Wiesbaden, 1901.—*Hebner.* Lehrbuch der Kinderkrankheiten. Leipzig, 1903.—*Neumann.* Säuglingsskorbut (Barlowsche Krankheit). Deutsche Klinik, Bd. 7, 1904, p. 341.—*Schuchardt.* Die Krankheiten der Knochen und Gelenke. Deutsche Chir., 1899.—*Stöltzner.* Pathologie und Therapie der Rachitis. Berlin, 1904.—*Zappert.* Rachitis. Deutsche

Klinik, Bd. 7, 1904, p. 427.—*Zesas.* Altes und Neues über Rhachitis. Zentralbl. f. Grenzgebiete. Sammelref., 1906, p. 321.—*Ziegler.* Ueber Osteotabes infantum und Rachitis. Zentralbl. f. Pathol., 1901, p. 865.—*Zweifel.* Aetiologie, Prophylaxis und Therapie der Rhachitis. Leipzig, 1900.

(e) OSTEOMALACIA

Definition.—Osteomalacia, like rickets, is a disease in which there is a general involvement of the bones. Osteomalacia, however, is characterized by regressive changes occurring in fully developed, strong bones of adults, resulting in softening, while in rickets the pathological changes involve the young, developing bone which, as a result, remains soft and does not become hard until later in life.

Etiology.—This rare disease occurs only in adults, most frequently of the female sex (only eight per cent of the cases reported were in males), who live among poor hygienic surroundings and have passed through a number of pregnancies. Its common occurrence in certain regions (South Germany, Italy, Switzerland) suggests some endemic influence; the marked hyperæmia of the medulla in the beginning of the diseases some inflammatory or vasomotor disturbance. Nothing definite is known, however, concerning the cause of the disease.

Pathology.—The pathological changes consist principally of decalcification (halisteresis). The changes begin upon the trabeculæ of the spongy bone and about the Haversian canals. Gradually the bones soften as the ground substance becomes decalcified and disappears. In the beginning of the disease hæmorrhages occur into the marrow, and later accumulations of pigment, the remains of the hæmorrhages, are found. The marrow becomes gelatinous and liquefied to form cysts. The new bone which develops in some areas, and which after fractures may lead to excessive callus formation, consists almost entirely of osteoid tissue, as calcification does not occur.

As a result of these changes the bones become fragile and so soft that they bend like india rubber when subjected to pressure or acted upon by the muscles. They become so soft that they may be easily cut with a knife or compressed between the hands. In the severest cases the shaft of the long bones may become as thin as paper or may completely disappear, leaving only the periosteum, which then resembles an intestinal loop, filled with pathological masses of bone marrow.

Clinical Course.—Slowly this frightful disease pursues its course, often interrupted by periods in which the changes remain stationary and improvement occurs. Occasionally the bones become sclerotic and the newly formed bone becomes calcified, and recovery occurs. After years of suffering, death from cachexia, pulmonary or intestinal diseases or from some terminal infection occurs.

The *puerperal form* begins during pregnancy, sometimes during the puerperium, with severe rheumatic pains in the lower part of the trunk. Gradually softening of the bones, which usually begins in those of the pelvis, is indicated by limitation of motion at the hip joint, giving rise to a peculiar waddling gait, and by a distinct decrease in the body length, the trochanters being forced upward as the necks of the femora become soft and yielding, the acetabula being pressed inward and backward and the promontory of the sacrum sinking downward and forward. The pelvis then becomes of a triradiate or clover-leaf shape. Often the disease is limited to the bones primarily involved, and in mild cases they may become sclerotic, and recovery occurs. But the reverse may happen if pregnancy occurs, the disease extending rapidly. If the softening extends to the vertebræ, ribs, and sternum, the chest wall becomes flattened and deformed and different varieties of spinal curvature will develop. The bones of the extremities may then become tender and soften. When the softening is complete the bones may be bent in almost any direction, while active motions become impossible. Only the bones of the skull and face are spared by these grosser changes. The patient, completely bedridden, dies in a few years from exhaustion or some disease of the lungs. The deformity of the chest predisposes to diseases of the lungs.

The *non-puerperal* cases, which almost always pursue a rapid course, usually begin in the vertebræ. They may begin with repeated pathological fractures (Riedel).

Diagnosis.—The diagnosis is possible only when the symptoms are pronounced. In the earlier stages, when the symptoms point to osteomalacia, X-ray pictures should be taken.

Treatment.—Phosphorus (combined with cod-liver oil) should be administered for a long time, as hardening of bone, which in the mild puerperal cases may occur spontaneously and end in recovery, is favored by this drug. The general strength of the patient should be improved by good nutritious diet and hygienic conditions. The deformed extremities should be maintained in proper position, if necessary, by extension dressings.

If pregnancy occurs, the induction of premature labor is advised by some, not only for the purpose of checking the disease, but also for the purpose of obviating such operations as craniotomy, cephalotripsy, and Cæsarean section. The removal of the uterus and its appendages, according to Porro, or of the ovaries alone, according to Fehling, has been followed in many cases by rapid improvement and recovery.

Paget's Disease of Bone.—Paget's disease of bone, or *osteitis deformans* (osteomalacia chronica deformans hypertrophica, osteomyelitis fibrosa), is closely related to osteomalacia. It occurs most frequently in

adults of an advanced age, and affects usually the shafts of the bones of the lower extremity (frequently the tibia on one side), the clavicles, the calvarium, the ribs, and the vertebræ. Deformities, accompanied by considerable painful thickening of the bones, gradually develop. There is a decrease in body length, as the vertebræ sink together and the bones of the lower extremities bow. The arms (which remain normal) appear long, and the general appearance of the patient, with the clumsy gait, the drooping shoulders, the flexed head, the prominent abdomen, and with the lower extremities slightly bowed and rotated outward, remind one of a large anthropoid ape (*vide* Schuchardt). Death, which follows after years of suffering, is due most frequently to some disease of the lungs, the development of which is favored by the narrowing of the thorax. It is also relatively frequently caused by sarcomas which develop from one or many of the diseased bones.

Pathology.—According to von Recklinghausen and Stilling, the pathological changes in this disease, concerning the cause of which nothing is known, consist of an atrophy of the bone by decalcification and lacunar resorption, combined with the formation of large amounts of osteoid tissue and the transformation of the marrow into a connective tissue poor in cells. Multiple cysts and giant-celled sarcomas (tumor-forming osteitis deformans of von Recklinghausen) may develop in the diseased bones.

If *tumor-forming osteitis deformans* is limited to a single bone it may be difficult to make a diagnosis. It is less difficult to differentiate between it and giant-celled sarcomas, which may be multiple and cystic (Haberer), than between it and multiple bone cysts. The latter develop in young people in the metaphyses of long bones, especially in the femur and humerus, appearing as unilocular, sometimes as multilocular, cystic fibrocartilaginous tumors which gradually expand and destroy the bone. They develop slowly after insignificant injuries, and attention is often first directed to them by spontaneous fractures. The majority of these cysts are not to be regarded as due to a local limited osteodystrophy (von Mikulicz), but as cystic endochondrofibromas, and should be classified with tumors. The cysts which occur in *osteitis serosa* and echinococcus cysts should be thought of in making a differential diagnosis.

Osteitis deformans may easily be confused with the inflammatory bone thickenings associated with chronic suppurative and syphilitic osteitis. This mistake may be avoided by taking an X-ray picture, which indicates the absence of or diminished amounts of calcium salts, and by carefully observing the clinical course of the disease. Osteitis deformans differs from leontiasis ossea in that in the former the calvarium is thickened while the base of the skull and the bones of the face are spared.

Treatment is of no avail. The extremities become so heavy that a supporting apparatus cannot be worn. The disease progresses steadily, and as there is but little tendency to consolidation, even osteotomy for the correction of deformities has been given up (Schuchardt, Schmieden). If the disease is limited to one of the extremities, amputation is indicated (Schmieden).

(f) BONE DISEASE OCCURRING IN MOTHER-OF-PEARL WORKERS

According to Englisch and Gussenbauer, elastic swellings which develop suddenly with pain in different bones are observed in young people who work with mother-of-pearl. A circumscribed bony thickening remains after the swellings subside. The swellings usually develop upon the metaphyses of long, hollow bones, but may extend from here to the epiphysis or diaphysis. They also occur upon the short and flat bones. Acute exacerbations may develop, and then the thickenings upon the different bones enlarge. There is no suppuration, necrosis, or disturbances of the general condition. The swellings cause but little trouble and may subside spontaneously if the patient stops his work.

The frequent occurrence of the swellings about the vascular metaphysis (cf. X-ray pictures of injected bone, pp. 417–418), and Gussenbauer's demonstration of dust of mother-of-pearl in the lungs of dogs after inhalation experiments, justify the view advanced by Englisch and Gussenbauer that the inflammatory changes follow the hæmatogenous deposition of particles of pearl dust or its organic constituent (conchiolin) in the bones. Broca and Tridon have made the only pathological investigations. They merely found evidences of a rarefying osteitis in pieces of bone which had been chiseled away.

It is possible to confuse this disease with the subacute forms of suppurative osteomyelitis.

It is not necessary to institute any special line of treatment, as the lesions heal spontaneously when the patient changes his occupation.

LITERATURE.—*Broca* und *Tridon.* Ostéomyélite des nacriers (Conchiolin-ostitis). Revue de chir, 1903.—*Gussenbauer.* Die Knochenentzündungen der Perlmutterdrechsler. Arch. f. klin. Chir., Bd. 18, 1875, p. 660.—*Haberer.* Zur Kasuistik der Knochenzysten. Arch. f. klin. Chir., Bd. 76, 1905, p. 559.—*Heineke.* Ein Fall von multiplen Knochenzysten. Beitr. z. klin. Chir., Bd. 40, 1903, p. 481.—*Küster.* Ueber fibröse Ostitis mit Demonstration. Chir. Kongr.-Verhandl., 1897, II, p. 333 und Disk. Schlange I, p. 134.—*v. Mikulicz.* Osteodystrophia cystica. Zentralbl. f. Chir., 1904, p. 1323.—*Schmieden.* Beitrag zur Kenntnis der Osteomalacia chronica deformans hypertrophica (Paget). Deutsche Zeitschr. f. Chir., 1904, Bd. 70, p. 207.—*M. B. Schmidt.* Osteitis deformans. Ergebnisse d. allg. Path. von Lubarsch u. Ostertag. 5. Jahrg., 1900, p. 949.—*Schuchardt.* Die Krankheiten der Knochen und Gelenke. Deutsche Chir., 1899, pp. 114–132, 149.

PART VI

TUMORS

I. GENERAL PART

CHAPTER I

DEFINITION AND CLASSIFICATION

Definition.—By the terms true or autonomous tumor, new growth, neoplasm, and blastoma is not to be understood every cellular proliferation resulting in the enlargement of the tissues or organs involved, but only those new formations of tissues which have very definite characteristics, among which the following are the most important:

1. The development of a true tumor is entirely independent of the organism, except that it receives nutrition from it. The growth fulfills no physiological function.

2. The structure of the tumor varies more or less from that of the surrounding normal tissues, and is atypical even from the first beginnings of its development.

3. A tumor, as a rule, has no typical limits of growth. There is always the tendency to progressive growth, notwithstanding the fact that there may be long periods in which the tumor remains stationary or even temporarily decreases in size. A permanent cessation of growth is observed in many benign tumors. Spontaneous involution is rare. Of course necrosis, followed by disappearance of the tumor, cannot be regarded as involution strictly speaking.

4. The causes and the nature of tumor formation are unknown. All hypertrophies and hyperplasias, inflammatory swellings, and infectious granulomas (occurring in tuberculosis, actinomycosis, glanders, leprosy, syphilis, rhinoscleroma) are to be distinguished from true tumors. Usually only the connective tissues are involved in the inflammatory growth, and the tissues which are formed do not differ greatly from the normal. The embryonal character of the tissue in inflammatory growths is maintained and cicatrization prevented by some definite cause (irritation of all kinds or bacterial infections), and when the cause is removed

the embryonal tissues become transformed into tissues of the adult type. Yet there are tumors, as Virchow long ago emphasized, that can scarcely be differentiated from hyperplastic new growths of an inflammatory nature, and the cells composing them scarcely exceed the limits of normal growth.

Cysts.—Sacs filled with fluid or caseous contents should only be classified with true tumors when they have developed from solid tumors (degeneration cysts, cystadenoma) or when the tissues comprising their walls have proliferated and have assumed the characteristics of new growths (epithelial, dermoid, and teratoid cysts).

Classification.—The classification of tumors has been based, since Virchow's classification, upon their histologic and histogenetic relations. According to Ribbert—whose classification is followed in greater part, for a classification based upon a clinical view-point alone is impossible—tumors may be divided into the six following groups:

I. Tumors developing from connective tissues.
   1. The fibroma (composed of fibrous tissue).
   2. The lipoma (composed of fat).
   3. The chondroma (composed of cartilage).
   4. The osteoma (composed of bone).
   5. The angioma (composed of blood vessels).
   6. The sarcoma (composed of unripe connective tissues, rich in cells) with its subdivisions:
      (*a*) Composed of cells of any of the connective tissues.
      (*b*) Composed of cells resembling lymphoid cells.
      (*c*) Composed of mucoid tissue—*myxoma, myxosarcoma.*
      (*d*) Composed of pigment cells—*melanoma, chromatophoroma.*

II. Tumors developing from muscular tissue.
   1. The rhabdomyoma (composed of striated muscle).
   2. The leiomyoma (composed of smooth muscle).

III. Tumors composed of nervous tissue.
   1. The neuroma (developing from nerves and ganglion cells).
   2. The glioma (developing from glia cells).

IV. Tumors developing from epithelium.
   1. The fibro-epithelial tumors.
   2. The carcinoma with its subdivisions depending upon the origin of the epithelium.

V. Tumors composed of endothelium.
   The endothelioma.

VI. Mixed tumors.

According to Virchow's classification, which is still valued highly, there are *histioid tumors*, the structure of which resembles closely the structure of normal tissues (e. g., fibroma, lipoma, osteoma); *organoid tumors*, which resemble in structure the different viscera of the body, being composed of an interstitial tissue and a parenchyma; and *systemadoid* or *teratoid tumors*, with a complicated organic structure resembling that of the body or a part of it.

CHAPTER II

ETIOLOGY OF TUMORS

THE etiology of tumor formation is still obscure. It is known that there are a number of conditions which favor the development of tumors, but no one alone is sufficient to explain the beginning of a new growth. Naturally, as the etiology is obscure, the greatest importance has been attached to the conditions which apparently favor growth.

1. **The Theory of Fœtal Residues or Embryonic Rests.**—[" This theory originated in Virchow's suggestion that in ossification of cartilage small islets might be left, which subsequently grow and develop into enchondromas; the idea was at a later date expounded by Cohnheim to include all tumors."—Rose and Carless, " Manual of Surgery," p. 177.]

According to this theory, tumors develop from germinal tissue which has been displaced or separated from its normal connection during embryonic development, or from tissues which ordinarily undergo involution, but have maintained their embryonal characteristics; in a certain sense, then, from local tissue malformations (Cohnheim, Ziegler). Epidermoids and dermoids develop from displaced ectoderm; entero-cysts from displaced germinal mucous membrane; tumors from displaced adrenal, thyroid, and mammary rests, from branchial clefts, the urachus and vitelline duct; true mixed and teratoid tumors from dormant displaced embryonal tissue. To these may be added Virchow's heterologous tumors, which undoubtedly develop from displaced germinal tissue. They differ from the homologous tumors in that they develop in tissue or organs to which they bear no resemblance histologically (e. g., lipoma of the pia mater and brain, myoma within the kidney, chondroma in the viscera, and tumors developing from congenital anomalies such as pigmented moles of the skin). Finally, it is not to be doubted that a number of tumors, such as angiomas, lipomas, fibromas, chondromas, myomas, sarcomas, which are often congenital and associated with all sorts of malformations develop from embryonic rests.

2. **Relation Between Inflammation and Trauma and Tumor Formation.**—That inflammation and trauma not infrequently precede tumor formation is well known to every clinician. It is not known, however, in what way the inflammation or trauma stimulates the tissues to atypical proliferation.

Tissue changes resulting from chronic irritation of different sorts and from chronic inflammation prepare the way for the development of tumors. It is well known that the chronic eczema developing in workers in paraffin and coal-tar, the inflammatory condition of the scrotum in chimney-sweeps, the chronic balanitis in phimosis, and the chronic dermatitis of the hands in X-ray workers favor the development of carcinoma. Besides, there is a tendency for carcinomas to develop in chronic ulcers, such as varicose ulcers of the leg and chronic ulcers of the stomach, in traumatic ulcers of the tongue and cheek (produced by carious teeth), in tuberculous and syphilitic ulcers of the skin, in chronic fistulæ following necrosis of bone or developing about the rectum, and in scars resulting from the healing of chronic ulcers. Hyperplastic new growths, such as leucoplakia, hyperkeratosis, papillary warts, mucous polypi forming upon an inflammatory base, not infrequently develop into carcinoma, occasionally into sarcoma or melanoma.

The causal relationship between pipe smoking and shaving and carcinoma of the lip, between pressure upon a definite area and the development of lipomas, etc., is less obscure than that between new growths and the conditions above mentioned.

A single trauma may be regarded as the cause of a tumor only in case of epithelial cysts in the palm of the hand. These cysts develop from pieces of skin which have been displaced and carried into the deeper tissues in punctured, incised, and gunshot wounds.

In all other cases it is impossible to demonstrate a direct relationship between trauma and tumor formation. It is possible that when a sarcoma develops at the seat of a fracture, a carcinoma of the stomach after a contusion of that abdominal wall, that the tumor was already present at the time of the injury, but had given rise to no symptoms, and that attention was drawn to it by the injury.

Statistics concerning the relationship between trauma and tumor formation are not convincing, as the tendency is too deeply rooted in the human mind to associate a local ailment with a local cause (Cohnheim).

It may, however, be regarded as certain that trauma and chronic inflammation, stimulating as they do the regenerative activity of the tissues, exercise a marked influence upon the growth of preëxisting tumors. This is an important point in accident insurance, as the company can be held liable only when a direct relationship between the

accident and the lesion can be demonstrated. It is well known how much more rapidly a malignant tumor grows after incomplete excision or cauterization. In the different statistics the causal relationship between trauma and tumor formation varies from 2.5 to 44.7 per cent (Borst).

3. **Heredity and Congenital Predisposition.**—Heredity or congenital predisposition seems to be an etiological factor in a certain number of cases. It seems to play a part in the development of some nævi, angiomas, multiple fibromas of the skin and nerves, lipomas, enchondromas, and exostoses which are either present at birth or develop later in life. It is remarkable that tumors not infrequently develop in the members of the same family, even in the same organs. One then speaks of a local predisposition of certain parts of the body or of certain organs. In carcinoma, according to Roger Williams, heredity, which may even extend to the part involved, may be established in 10.5 per cent of the cases (Lücke and Zahn).

The different views concerning the development of tumors may at the present time be divided into two groups. To the first group belong the hypotheses of Cohnheim and Ribbert, according to which tumors develop from cells which have been separated from their organic connections and displaced either during embryonal or extrauterine life. In the second group may be combined all those hypotheses according to which tumors develop from cells normally placed, but which have acquired the property of unrestrained growth as the result of the action of some unknown influences (irritation of different sorts) (Virchow) or perhaps even of parasites.

4. **Cohnheim's Theory.**—According to Cohnheim, all true tumors develop from superfluous, misplaced, or abnormally persisting centers of embryonic tissue which may be stimulated to growth by a number of different causes (increased nutrition, decrease of resistance to growth, physiological increase or decrease of local or general growth, Borst).

Dermoids and teratomas, tumors of accessory organs and displaced adrenal rests, cysts arising from the branchial clefts, the urachus, vitelline and thyreoglossal ducts, which undoubtedly develop from displaced or non-involuted embryonal tissue, support this theory.

Cohnheim's theory, however, lacks anatomical foundation, and there are a number of facts which make it impossible to apply it to the development of tumors in general. In the first place it is scarcely possible to conceive that tumors developing in advanced life have sprung from displaced tissues which have maintained their embryonal characteristics through all the preceding years. Besides the transplantation of embryonal tissues into animals of the same species has never been followed by tumor formation, the embryonal tissue, if it has remained

alive and has developed, becoming transformed into tissues resembling more or less closely those of the adult type (Zahn, Leopold, Birch-Hirschfeld and Garten, Féré, Schmieden, Wilms, and others).

5. **Ribbert's Theory.**—According to Ribbert, true tumors develop from germinal tissue which is either displaced during development or later in life by traumatism or after inflammatory processes. When displaced this germinal tissue is separated from its normal physiological connections and becomes an independent center of growth. Ribbert believes that the independence of this tissue explains satisfactorily its unlimited growth, for the tension of the tissues no longer exercises a restraining influence, as the normal tissues do. Inflammatory hyperæmia, and the hyperæmia associated with different forms of trauma, favor rapid and excessive proliferation.

The development of epithelial cysts, such as occur in the palm of the hand, supports Ribbert's theory of the post-embryonal separation of groups of cells from their organic connections. These small cysts develop from pieces of epidermis which are carried beneath the cutis by foreign bodies or by injuries. The separation and displacement of groups of cells can be frequently demonstrated in chronic inflammatory processes. The composition of the germinal tissue determines the character of the tumor; for example, a fibroma develops from displaced fibrous tissue, a lipoma from fatty tissue, a carcinoma from epithelium. A sarcoma develops if the germinal connective-tissue matrix remains of an embryonal type.

This theory, like Cohnheim's, attempts to place tumor formation upon a single, definite basis. It has gained more and more recognition of late. It, however, also presupposes the action of some unknown influence, for apparently the displaced cells are incapable of spontaneous proliferation, and it has been established by a number of experiments that tumors do not develop after the transplantation of tissue of various kinds. Besides, in many diseases cellular and tissue emboli (bone marrow and giant-cells, fat, liver, and placental cells, and chorionic villi) are deposited in viscera and other tissues without giving rise to tumor formations. Ehrlich's work throws some light upon the nature of the unknown influence which prevents proliferation in these cases. According to Ehrlich's theory, the organism possesses a certain protective mechanism (atreptic immunity) which prevents abnormal growth. When the immunity is decreased the tumor germs may take from the body the food-stuffs required for proliferation.

According to Hauser, tumor formation may begin in cells normally placed. But in this theory it must be presupposed that there are certain special biological changes in the cells which lead to tumor formation. It may be assumed, for example, that the normal cells, which

may be stimulated to increased proliferation by all sorts of irritation, gain an increased energy for growth while their functional activity is decreased (Beneke, Hauser, Lubarsch, O. Israel), or that they assume toxic properties (Marchand) which destroy adjacent tissues and render an infiltrating growth possible. Thiersch attempted to explain the development of carcinomas by supposing that the connective tissues undergo a certain atrophy, associated with a relaxation of their strata, and that they then no longer oppose a barrier to the epithelium still possessed of its full power of reproduction. Von Hansemann presupposes in malignant tumors an anaplasia of the cells from which they develop, and therefore the tumor remains of a primitive structure as the anaplastic cells are not capable of differentiation into tissues of an adult type. According to this theory, anaplastic cells respond to stimulation by developing into malignant tumors, while normal cells form merely hyperplasias in the broadest sense of the word. It is a fact that tumor cells are very primitive in structure, resembling closely the embryonal prototypes of the tissue from which they spring; for example, large connective-tissue cells which do not form intercellular fibrillæ are found in sarcomas; epithelial cells which do not cornify and do not secrete are found in many carcinomas of the skin and mucous membrane respectively. Ribbert does not consider this return of cells from an adult, and differentiated to a primitive and simple type as necessary for tumor formation, but merely as a factor which favors growth when tumors develop from fully differentiated elements.

6. **Parasitic Theory.**—Bacteria which have been found in tumors have been shown to be merely harmless saphrophytes and not the essential cause. Critical examination has shown that the blastomyces, protozoa, rhizopoda, infusoria, and sporozoa (among the latter coccidia, gregarinæ, plasmodia, psorospermia) which have been described in tumors were altered tumor cells undergoing regressive changes, such as vacuole, keratohyalin, and colloid formation, or cell inclusions, consisting in part of tumor cells, in part of degenerated leucocytes or epithelium (*vide* Borst). L. Pfeiffer and Adamkiewicz regard the carcinoma cells proper as the parasites.

A number of different investigators (Busse, Jürgens, Schüller, Sjöbring, and others) have been successful in cultivating parasites from fresh tumor tissue, but they have never been able to produce by inoculation any changes which could be regarded other than of an inflammatory nature.

Only the transplantation of living tumor tissue—for example, the transplantation of carcinomatous tissue from one to another part of a patient suffering from the disease (Hahn, von Bergmann, Cornil), or of tumor tissue from one animal to another animal of the same species

(dog, rat, mouse) (Novinsky, Weber, Hanau, von Eiselsberg, Geissler, Moran, Jensen and others)—has led to any definite results.

The experiments made by Gaylord, of the New York State Cancer Laboratory, and by Ehrlich have given the most important results, which are also of significance in another direction. These investigators have been able, by carrying inoculations through a number of mice, to increase the power of growth or virulence of the tumor masses, just as the virulence of bacteria is increased by passing them through animals, so that finally almost all inoculations with the most virulent material are successful. Sometimes in these experiments a carcinoma becomes transformed into tissue resembling that of a sarcoma or into pure forms of sarcomas, as the epithelium is finally suppressed by the more rapidly proliferating stroma. As regards the parasitic theory, all these findings (similar to the so-called inoculation recurrences in the scar after operations for carcinoma) merely show that encapsulated, well-nourished tumor cells may develop in other parts of the body, being similar, therefore, to metastatic growths. Transplantation experiments also show that the tumor cells may retain their growth energy, not, however, that a parasite has been inoculated with the tumor tissue and has caused the development of a new growth.

From a clinical view-point, practically, only facts which relate to carcinoma have been employed to sustain the parasitic theory of the origin of tumors (Czerny); such as, that carcinomas develop upon parts most frequently exposed to external influences (face, neck, hands); are most common where wounds are common, and apparently provide infection atria (ulcers of all sorts, fistulæ, eczema, scars resulting from wounds or ulcers, fissured nipples, erosions of the cervix); or where there are changes resulting from chronic irritation (chronic inflammation of the skin, hyperkeratosis, seborrhœa, eczema, leucoplakia, chronic balanitis in phimosis). The frequent involvement of those parts of the gastro-intestinal tract most often exposed to traumatic and inflammatory irritation (margin of the tongue injured by carious teeth, œsophagus, cardia, pylorus, flexures of the large intestines, rectum) also supports this theory. Uncleanliness seems to play a rôle and to speak for a parasitic cause (frequent occurrence of carcinoma of the face among the poorer classes, of cancer of the mouth when the teeth are badly cared for, of carcinoma of the breast when the nipples are dirty and scaly). The occurrence of multiple carcinomas in the gastro-intestinal tract; the few cases of so-called implantation carcinomas, for example, implantation from the tongue to the mucous membranes of the cheek lying opposite (Lücke), from the lower to the upper lip (von Bergmann), from one peritoneal surface to the opposite (Beneke); and the occurrence of carcinoma in many members of the same family or in a

number of families living in the same house or neighborhood have been supposed by many to speak for a parasitic origin. Broca has carefully reported the history of a family in which sixteen out of twenty-six members, representing three generations, were afflicted with carcinoma.

Objections to the Parasitic Theory.—There are a number of objections which may be raised against the parasitic theory, and even the frequent involvement of the parts above mentioned may be satisfactorily explained without resorting to it. In the first place, certain types of tissue are always reproduced in the different tumors, even in the different varieties of carcinomas. How would it be possible that in a parasitic infection only one definite form of cell is always stimulated to proliferation (for example, only the epithelium), while the connective, endothelial, and glandular tissues are acted upon by the infection at the same time? In this case there must be at least as many varieties of parasites as there are varieties and sub-varieties of tumors, leaving out of consideration mixed tumors, the complicated structure of which alone speaks against a parasitic origin. The development of metastases, composed of cells resembling those of the primary growth, and the growth of the tumor without stimulating the surrounding tissues to proliferation are weighty arguments against the theory (cf. Borst, Ribbert).

CHAPTER III

FORM, GROWTH, AND CLINICAL SIGNIFICANCE OF TUMORS

Different Forms which Tumors may Assume.—Among the many forms which tumors situated superficially or deeply may assume, the round, nodular form is the most common. As a tumor develops it may change into a tuberculated, bulbous mass, the form being influenced by anatomical relations. The following forms of tumors are differentiated upon the surface of the skin and mucous membranes: Tumors with broad and thin pedicles; fungoid, pendulous, verrucous, villous, papillary tumors with numerous thornlike elevations; and cauliflowerlike growths with a dendritic arrangement of the proliferating tissues. Several different forms may be combined in the same tumor.

Expansive and Infiltrating Growth.—A tumor, as it grows, pushes aside or infiltrates the surrounding tissue, the former being known as *expansive*, the latter as *infiltrating growth*. The increase in the size of a tumor is due to the proliferation of its constituent parts alone, and not to the transformation of the infiltrated tissue into tumor cells and their proliferation to form tumor masses, as was formerly con-

sidered to be the case. A tumor growing by expansion has sharp boundaries, may easily be separated from the surrounding tissues, and has a distinct capsule formed by the thickening and reactive proliferation of the surrounding tissues; while an infiltrating tumor has more or less indistinct boundaries and is intimately attached to the surrounding tissue. Sometimes a growth which is expansive in the beginning later becomes infiltrating.

A tumor is always nourished by blood vessels, which enter it from the surrounding tissues. In slowly growing tumors the blood supply is usually sufficient to nourish the entire tumor, while in rapidly growing tumors it is often insufficient, and parts of the tumor may become necrotic during its later development. Frequently the necrosis is preceded by fatty and mucoid degeneration. Often tumors, especially those with long pedicles, become œdematous as the result of venous stasis. When stasis occurs, fluids may be pressed from the cut surface of the tumor as from a sponge. It then resembles closely myxomatous tissue.

Regressive Changes.—Regressive changes occur when the infiltrating growth invades and occludes the blood vessels. The regressive changes lead to the formation of cavities in the interior of tumors, of ulcers upon the surface, and not infrequently to contraction of the connective tissues.

Clinical Significance.—The clinical significance of tumors rests in the first place upon the harm they do, which may be the direct result of their enlargement and other properties of tumor tissue. In expansive growths the amount of harm done the organism depends entirely upon the importance of the structure or of the organ pressed upon or displaced. For example, pressure upon large vessels causes circulatory disturbances; upon nerves and the spinal cord, irritation or paralysis; upon the brain, severe symptoms or death; while a tumor upon the surface of the body, even if very large, may cause but slight inconvenience. Infiltrating growths do much more harm, as they press upon the infiltrated tissue, which is destroyed (e. g., destruction of an entire viscus and replacement by tumor masses, erosion of large vessels).

The harmful effects of tumors may also be due to recurrences, metastases, and the so-called cachexia which they induce.

Recurrence of Tumors.—Recurrences occur after removal only when part of the tumor tissues has been left. Naturally this is much more frequent when tumors infiltrate the surrounding tissues than when they are circumscribed and encapsulated. The recurrences may develop in the area from which the tumor was removed, or, if the cells have been carried by the lymph stream, in surrounding structures.

Metastatic Growths.—By metastatic tumors are understood those developing secondary to the primary growth in distant parts of the

body. They develop from tumor tissue (cells, groups of cells, or pieces of tumor tissue) which has been carried by the lymphatic vessels and blood vessels to distant parts, after one or the other of these has been invaded by the infiltrating growth. The cells carried by the lymph stream are arrested in the adjacent lymph nodes and develop into secondary tumors. If the tumor cells pass into blood vessels, as is frequently the case in advanced carcinomas and sarcomas, or into the thoracic duct from some of the smaller lymphatic vessels, they are distributed and deposited in different parts of the body in the form of emboli (hæmatogenous metastases).

If the tumor invades the veins of the systemic circulation, tumor masses may be carried by the blood stream into the lungs. If the emboli are small enough to pass through the capillaries of the lung (6 μ in width), they are carried to the left heart, and from here into the arterial system, to lodge where there is a hyperæmia or where the capillaries are very narrow (liver, kidney, more rarely other viscera, bone, skin). Embolism of one of the larger branches of the pulmonary artery may cause immediate death. If the tumor tissue gains access to the portal vein, it will first be deposited in the liver. It cannot be estimated how many tumor cells die in the lymph and blood stream or fail to develop after they are deposited. It is not to be doubted that degenerating non-viable cells as well as viable cells are carried in the emboli, and that all of them are not able to form metastases. More rarely the following varieties of extension occur by way of the lymphatic and blood vessels: (1) Continuous extension by growth of the tumor elements within the lumen of the vessel (e. g., a carcinoma grows for some distance in a lymphatic vessel to an adjacent node, extension of carcinomas of the stomach and intestines into radicles of the portal vein, and of a sarcoma or hypernephroma of the kidney through the renal vein into the inferior vena cava, and from here into the right heart); (2) retrograde extension, the tumor tissue developing against the current of the vessel involved. This may be the case if there is a marked venous stasis and the pulsation transmitted to the veins is more powerful than the blood current (Ribbert).

The occlusion of one of the principal lymphatic channels may so change the direction of the lymph stream that tumor cells may be carried in a direction opposite to that in which the stream normally flows in vessels entering into the collateral circulation. A continuous growth within the lymphatics extending to adjacent nodes is more frequent than the retrograde embolism above mentioned.

In serous cavities tumor cells may be disseminated upon the surfaces of the peritoneum, pleura, and pericardium by the movements of the viscera. Tumor cells may also be transplanted into an operation-wound

during the removal of malignant growths (inoculation metastases or recurrences). The structure of the secondary growth is always the same as that of the primary tumor.

The formation of metastatic growths is peculiar to tumors with an infiltrating growth. Tumors with an expansive growth do not invade lymphatic and blood vessels. According to Ribbert, the clinically significant but not sharp division of tumors into the benign and malignant is based upon this difference in growth, and not alone upon the characteristics of the tumor cells. Benign tumors, as a rule, grow slowly by expansion without forming metastases, and becomes dangerous only when they attain great size or interfere with the function of some of the important organs. Malignant tumors form metastatic growths giving rise to regional or general metastatic growths, and as they infiltrate tissues they destroy the tissues or organs involved. The more closely the cells composing a tumor approach an embryonal type, the more rapid the growth of the tumor and the more frequent the infiltrating growth will be.

General Constitutional Effects of Tumors.—Tumors may produce general constitutional effects which consist most frequently of a marked falling off in the nutrition of the body—the so-called *cachexia* of tumors. This cachexia is most pronounced in malignant tumors when accompanied by metastatic growths, but may also occur in benign growths if they are multiple (e. g., multiple lipomas) or if they attain an extraordinary size (e. g., fibromyoma of the uterus, large fibrolipoma of the skin). There are a number of different causes of cachexia, such as interference with the function of the viscus involved, interference with the general functional activity and nutrition of the body, pain, loss of sleep, and a number of other things. Fever and the absorption of the products of decomposition from the tumor and of putrefactive products from ulcerated tumors are important factors in causing cachexia. Finally, regressive changes in a tumor may give rise to dangerous complications, such as aspiration pneumonia in carcinoma of the mouth or peritonitis after perforation of a carcinoma of the stomach.

CHAPTER IV

THE GENERAL DIAGNOSIS OF TUMORS

The diagnosis of a tumor—that is, determining whether the lesion is a true tumor (differentiating it from hyperplasias, inflammatory infiltrations, infectious granulomas and cysts) and determining the character of the tumor—is based upon the following:

I. The previous and present history. The time at which the tumor began to develop, the determining cause, the mode of growth, and the way in which the tumor has extended. The local and general subjective symptoms are also of importance.

II. Upon the physical findings (A) the local, (B) the general, combined with (C) consideration of what varieties of tumors are most frequent in the area or organ involved, and (D) special diagnostic methods.

A. In the local examination the position and peculiarities of the tumor should first be determined by inspection and palpation.

Inspection should determine:

1. The position and extent of the tumor in relation to anatomical structures (region of the body, contour of the bone, muscles, tendons);

2. The form (round, oval, irregular, flat, hemispherical, nodular, pedunculated, fungous, cauliflowerlike, papillary);

3. The size (compared with well-known objects such as a pea, cherry, walnut, hen's egg, child's head, or accurate measurements of its transverse and longitudinal diameters);

4. The surface;

(*a*) Covered with skin or mucous membrane (of normal appearance, permeated with dilated vessels, hyperæmic, pigmented, tense and shining, transparent);

(*b*) Sloughing, ulcerated (secreting, bleeding slightly, covered with crusts);

(*i*) Edges of the ulcer (forming an elevated wall or flat, sharply cut, excavated, or eroded, firmly attached to underlying tissues or undermined, hard, or soft);

(*ii*) Floor of the ulcer (flat, depressed, craterlike, or filled with growths, necrotic, uneven, smooth, or fissured);

(*iii*) Surrounding tissue (normal, raised by tumor masses or invaded by secondary nodules);

(*iv*) The margins (sharply defined or indistinct, circumscribed, or diffuse).

Palpation should determine:

1. The relation of the tumor to the tissues covering it (whether the skin covering it may be raised in folds as the healthy surrounding skin, whether the skin or mucous membrane covering the tumor can be displaced over it, whether muscle lies between the skin and the tumor. The latter is to be determined by lifting the muscle up—for example, by lifting the sterno-cleido-mastoid—or by testing the function of the muscle—for example, by testing the rigidity of the abdominal muscles, by elevation of the arm to prove whether a tumor is beneath the deltoid).

2. The characteristics of the surface of the tumor. It may be determined when the examination is made concerning the displaceability

of the tissues covering the tumor whether its surface is flat and smooth, nodular, lobulated, or irregular.

3. The boundaries of a tumor, whether they are distinct or indistinct, whether sharply defined against the surrounding tissues or whether they disappear indistinctly into the deeper parts—for example, below the jaw, at the mastoid process—or gradually fuse with the normal tissue.

4. The position of the tumor and its anatomical relations (after palpation of adjacent tendons, muscles, bones, and viscera).

5. The relation of the tumor to surrounding and subjacent tissues (whether it can be moved here and there with the skin and soft tissues, or is situated deeply upon bone, or is firmly attached to tendons or fascia; whether it moves with tendons, muscles, or with the liver during respiration).

6. The consistency of the tumor. The most important characteristic to be determined is whether the tumor is soft or hard.

(*a*) Only the experienced, not the beginner, can detect the finer differences, such as the difference between the hardness of cartilage and bone, and between the elastic consistency of a lipoma, and the sensation imparted by a soft or hard fibroma.

(*b*) Fluctuation. Hard as well as soft tumors may fluctuate, depending upon whether the capsule surrounding the fluid or liquefied masses of tumor tissue is tense or relaxed. A hard, fluctuating tumor is elastic. In soft tumors the capsule must be made tense by pressure before fluctuation can be elicited.

In firmly attached or slightly movable tumors fluctuation is elicited in the same way as in abscesses. The index finger of each hand should be placed upon the tumor opposite each other, first at a small, later at a greater, distance from each other. The fingers should be laid flat upon the tumor, and only in examining soft tumors should any great amount of pressure be exerted. The left index finger (inactive finger) is then held quiet and motionless, while pressure is made with the right (active finger) which is then quickly removed. The inactive finger will be elevated by the displaced fluid if the tumor fluctuates.

If the tumor is movable it is best to grasp it between the index finger and thumb of each hand. If pressure is then made with both fingers of the right hand, those of the left will be raised if fluid is present.

Some tumors (lipoma, myxoma) very frequently impart the sensation of indistinct or pseudo-fluctuation. [Mistakes are not infrequently made even by experts in determining fluctuation. I myself have made the diagnosis of fluid in a case of fatty tumor in the infraspinous fossa covered by the infraspinatus muscle.]

(*c*) The condition found in dermoid cysts and the so-called fæcal tumors (hard fæcal masses), in which the depression made by the finger remains and only disappears when pressure is made upon the opposite side of the tumor, is spoken of as a *doughy* or *kneadable* consistency.

(*d*) Pulsation is best elicited by placing the hand flat upon the tumor without exerting pressure (in certain hæmangiomas in the same way as in aneurysms). If pressure is made upon the principal artery supplying the tumor, pulsations cease.

(*e*) A tumor is spoken of as compressible when it diminishes in size under pressure and enlarges again when the pressure is removed (hæmangiomas and lymphangiomas).

(*f*) Thrills are often felt in pulsating tumors when the hand is placed upon them (*vide* hydatid fremitus in echinococcus cysts).

(*g*) Tumors covered by a thin shell of bone, when palpated, impart a parchmentlike sensation or crepitation.

B. The general examination begins with:

1. The palpation of neighboring lymph nodes, which in many malignant tumors are enlarged and indurated, then follows:

2. The examination of other similar growths, if present (multiple tumors), and the search for metastatic growths of the skin, large viscera, and bones (by percussion or palpation).

3. The special examination of different viscera and systems; for example, examination of the urine (for albumin, blood, also for sugar in supposed pancreatic tumor), of the fæces (for blood, and mucus in tumors of the intestine), of the gastric juice (in tumors of the stomach), of the function of the kidneys (the solids of the urine are estimated by determining the freezing points of the urine discharged from each kidney), of the nervous functions (in supposed tumors of the brain, spinal cord, and peripheral nerves), of the blood (to determine the relative proportion of the cells in diseases of the blood-forming organs, the spleen, lymph glands, and bone marrow).

4. The critical examination of the general condition (cardiac function, anæmia, digestion, physical and mental characteristics).

C. What tumors or tumorlike formations occur most frequently in the area or organ involved? The answer to this question often makes a definite diagnosis possible, even after the results of a most accurate examination have been insufficient. A few examples will render this statement clear.

A hard, indistinctly fluctuating, round tumor which is slightly movable upon the subjacent tissues and has no connection with tendons is situated just beneath the skin in the palm of the hand. A tumor with similar characteristics situated about the eye would be diagnosed at once as a dermoid cyst, as they frequently occur here. Not so, however, in

the palm of the hand. Dermoid cysts do not occur here, while epithelial cysts (which never occur about the eye) do.

In considering the diagnosis of some tumors, their connection with a nerve may be definitely established. The characteristics of the tumor alone, which is round, of average hardness, and not adherent to the skin or underlying tissues, will suggest the variety. The few varieties of the tumors, however, which develop upon the peripheral nerves limit the differential diagnosis to a few new growths, and one has only to determine whether the tumor has grown slowly or rapidly to decide whether it is a fibroma or sarcoma.

In making the diagnosis of the character of a tumor of the breast, the position is very important. A number of tumors and tumorlike hyperplasias develop in the breast which do not occur in other parts of the body. A lobulated, nodular, soft tumor, lying beneath normal skin and displaceable upon the underlying tissue is not to be diagnosed, as it might be in the back, as a lipoma. The relation of the tumor to the breast must first be determined. Often the entire breast or parts of it may be hypertrophied and cystic (mastitis chronica cystica), a condition which might easily be mistaken for a subcutaneous lipoma.

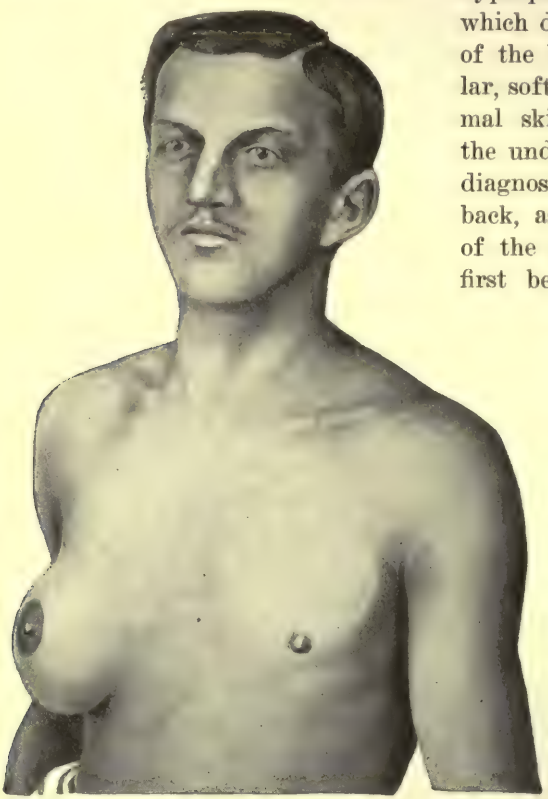


Fig. 282.—Gynæcomastia (Right Side), Male Patient Eighteen Years Old.

Tumors of similar characteristics occur even in the male breast and surrounding tissues. A similar lesion of the male breast may be a tumor or a condition known as gynæcomastia, in which the breast undergoes a hyperplasia of all its component parts (which may even be unilateral) and resembles the female breast (Fig. 282). Carcinoma of the breast has some special characteristics, such as very evident infiltration of the gland, retraction of the nipple, etc. Frequently

a definite diagnosis of carcinoma of the breast can be made much earlier than that of carcinoma of other parts.

Myelogenous and periosteal sarcomas, chondromas, and osteomas are the most common tumors of bone. In spite of this a flat or nodular, resistant, not sharply defined tumor which is attached to the bone, is covered by normal skin, and has grown rapidly cannot be diagnosed as a sarcoma without considering some other lesions. One should think of the enlargement associated with chronic suppurative and tuberculous osteomyelitis, and especially of a periosteal gumma, which occurs most commonly upon the diaphysis of the tibia, and search for other evidences or remains of the infectious diseases above mentioned should be made.

Any number of such examples might be cited. Those already mentioned show conclusively that an accurate knowledge of special pathology, vast experience in diagnosis, the ability to weigh possibilities, and an extended clinical experience are required before the diagnosis of tumors can be made correctly and with certainty.

Even the most expert diagnosticians are frequently unable to make a positive diagnosis, being unable to decide between a number of possibilities.

D. Special diagnostic aids. Aspiration, exploratory incision and excision with microscopical examination, X-ray pictures and special methods in abdominal tumors are diagnostic aids.

1. *Puncture and aspiration* of a swelling is important when it is difficult or impossible to determine its consistency (indistinct fluctuation). By inserting a needle or canula, it is possible to determine whether fluid is present or not, and if present the character of the same (serous, hæmorrhagic, purulent, mucoid, or fluid from echinococcus cysts).

2. The *exploratory incision* is sufficient in many cases to enable the surgeon to make a definite diagnosis as to the character of the tumor by the macroscopic appearance of its cut surface (e. g., lipoma or sarcoma, fibroma or carcinoma of the mammary gland). The harpooning of pieces of tumor tissue, which was employed extensively in preantiseptic times, is no longer practiced. Harpooning consisted of inserting an instrument provided in barbs (similar to a harpoon) and removing small pieces of tissue from the tumor, which could be used for microscopic examination (Middeldorpf's harpoon).

Excision of pieces of tissue for microscopic examination is often resorted to. Small pieces of tissue are removed for microscopic examination in cases in which it is probable that a tumor is malignant, but the symptoms are not pronounced enough to make a positive diagnosis possible. It is of most value when tumors are first beginning to develop (e. g., to diagnose between psoriasis, syphilis, and beginning carcinoma of the tongue, between carcinoma and papilloma of the larynx, in sus-

pected carcinoma of the uterus). A small wedge-shaped piece of tissue is removed from the surface of the tumor, and the resulting wound is touched with a thermocautery to control the hæmorrhage. In tumors of the larynx a good view of the tumor should first be had with the laryngoscope before the tissue is removed with forceps, in order to be certain that the tissue is removed from the tumor and not from the diseased area adjacent to it.

In many cases pieces which are separated and cast off from the tumor may be used for examination (e. g., tissue expectorated in tumors of the mediastinum and lungs, vomited in carcinoma of the stomach, passed in the urine in tumors of the bladder, in the fæces in carcinoma of the rectum).

X-ray pictures are of value in many cases. Hard tumors and those consisting of bone (osteoma, osteosarcoma), or containing pieces of bone (teratoma) may often be recognized in pictures by their nodular form and differentiated from inflammatory infiltrations and hyperostoses. The appearance of the bone from which the tumor develops depends upon whether or not it is destroyed by the new growth. Exostoses are attached by broad or thin pedicles to the surface of the bone. A periosteal sarcoma surrounds the bone which in the beginning at least is still normal, the contour of which can be seen through the shadow of the tumor. Small central tumors can be differentiated from inflammatory processes only when the cortical layer of bone covering them has become thinned. As a rule, bone surrounding inflammatory processes becomes thickened and sclerotic. Often it can be seen that the tumor has ruptured through the thinned and expanded parts of the cortex at a number of points. Tumors of the mediastinum can often be diagnosed by an X-ray examination.

In the examination of abdominal tumors a number of different diagnostic methods are employed to determine the position of the tumor and its relation to neighboring organs. For example, the stomach may be distended with an effervescing powder, the colon with air or water in order to determine the relation of the tumor to these organs, whether it is in front or behind or whether the tumor changes its position as the organs are distended.

EXAMPLE OF THE METHOD EMPLOYED IN MAKING THE DIAGNOSIS OF A TUMOR

LIPOMA

I. Anamnesis: Symmetrical, gradual growth for a number of years, no cause, no trouble, only discomfort because of size, general condition of patient unchanged.

II. Status: (A) The local examination by inspection:

1. Position and extent. A tumor is present upon the back of the patient, in the right scapular region, covering almost the entire bone.

2. Its form is almost oval; in profile hemispherical, with an indistinct tonguelike process upon the medial side.

3. Its size is a little larger than the head of a newborn child.

4. Its surface is covered by normal skin.

5. Its boundaries are sharp and distinct except along the lower part.

By palpation:

1. The skin may be raised in folds from the surface of the tumor, but the skin is thinner than that of the area adjacent. [In doing this an irregular wrinkling is produced by the trabeculæ of connective tissue which divide the lobules of the tumor.] In some areas the skin covering the tumor is less movable.

2. The surface of the tumor is smooth throughout. At the margins distinct, round projections may be felt, and upon the medial side a large process. Both may be made visible by rendering the skin tense.

3. The boundaries of the tumor are sharply defined and distinct from the surrounding structures except along the lower margin.

4. The position of the tumor corresponds to the right scapula. Its lower part disappears beneath the latissimus dorsi, the border of which may be distinctly palpated.

If the muscle is made to contract by pressing the arm against the side of the chest, the lower part of the tumor becomes harder and its lower boundary still more indistinct.

5. The tumor is but loosely attached to underlying structures (fascia), as it can be easily displaced in all directions.

6. Its consistency is soft. If the tumor is grasped with four fingers, indistinct or pseudo-fluctuation may be elicited.

(B) The general examination reveals no enlargement of the regional lymph nodes. Another tumor about as large as a walnut with similar characteristics is situated upon the outer side of the left thigh. The general appearance of the patient is good.

(C) The part involved (the back in the region of the shoulder) is a common site for subcutaneous lipomas. The findings correspond to such a tumor. Other tumors occurring in this region, such as a sarcoma, may be excluded because of the slow growth; atheromas because of their round form and small size.

(D) Special diagnostic aids, such as puncture and aspiration, are not necessary.

Diagnosis: Circumscribed subcutaneous lipomas over the right scapula and on outer surfaces of left thigh.

SARCOMA

I. Anamnesis: For one half year an enlargement in the region of the right scapula has been noticed. For two months the growth has been rapid, accompanied by pain and limitation of motion of the arm. For two weeks marked general weakness, attacks of coughing, and pain upon breathing in left side of thorax have been present.

II. Status: (A) Local examination by inspection:

1. Size and extent. A tumor in the region of the right scapula, involving the area below the spine, extending about 4 cm. below it.

2. Its form is that of a round, flat swelling.

3. Its size corresponds to that of a child's head.

4. Its surface is covered in the upper half by skin which appears normal, while that covering the lower half is traversed by dilated veins.

5. The boundaries of the tumor are ill-defined.

By palpation:

1. The relations of the tumor to structures covering it differ. The skin covering the upper part of the tumor may be raised in folds, and is of the same thickness as the surrounding skin. In the lower part the skin is so firmly attached to the surface of the tumor that it cannot be raised in folds.

2. The surface of the tumor is smooth with superficial furrows.

3. Its boundaries can be distinctly made out in the lower part only.

4. The position of the tumor corresponds to the right scapula. Above the boundary becomes indistinct at the spine of the scapula; medially and laterally it fuses with the neighboring muscles; below it extends two fingers' breadth beyond the angle of the scapula. There is no muscle between the tumor and the skin, at least muscle cannot be recognized during active movements. The tumor is attached to the scapula, following its movements.

5. It is also firmly attached to the underlying bone, as it cannot be displaced over the scapula.

6. The consistency of the tumor is hard, and only at a small point in the lower part where it is attached to the skin can fluctuation be elicited.

(B) The general examination reveals no enlargement of the axillary lymph nodes. Similar growths are not present upon any other part of the body. A general physical examination reveals some involvement of the left lung. There is a left pleuritic effusion and the expectoration is bloody. Aspiration reveals an effusion which is bloody in character. The sallow appearance of the patient and the great weakness are striking.

(C) The area involved is the favorite site for lipomas (see previous example), but a lipoma does not become attached to bone. Exostoses,

enchondromas, and sarcomas are the most common of the other varieties of tumors developing on the scapula. The indistinct boundaries and rapid growth speak for a sarcoma, and there are no findings which suggest that the tumor is not of this character. The involvement of the lung is to be regarded as metastatic in character. Hæmorrhagic pleural exudates are frequently associated with tumors of the lung.

(D) Aspiration of the small fluctuating area reveals dark blood. The X-ray pictures show the indistinct shadows of the scapula.

Diagnosis: Cystic osteosarcoma of the right scapula with metastatic growths in the lung.

TUMOR OF THE BREAST

I. Anamnesis: For six months a woman forty-five years of age has noticed a hard nodule in the left breast. This was first noted after a blow. For some weeks the skin covering the rapidly growing mass has been discolored. A short time before the present examination the skin covering the mass ulcerated and a hæmorrhagic fluid was discharged. The open area has enlarged rapidly and bleeds profusely at times. Severe pains radiate from the mass and a foul-smelling secretion is poured out. The strength of the patient has been considerably reduced since the lesion was first noticed.

II. Status: (A) Local examination by inspection:

1. Size and extent. The left breast is about twice its normal size, and upon the outer side the new growth extends beyond the limits of the breast.

2. The form of the enlarged breast is that of an irregular, nodular hemisphere.

3. Its size corresponds to that of a large normal mammary gland.

4. Its surface:

(*a*) In the upper half is covered by normal skin. The nipple is greatly retracted. The skin to the medial side of the nipple, covering the nodular growth, is of a bluish red color.

(*b*) To the outer side of the nipple in the lower half of the gland is an ulcer the size of a dollar which is surrounded by a bluish red zone. It pours out a foul-smelling, serohæmorrhagic secretion, and is covered with crusts and bleeds at some points.

(*i*) The edges of the ulcer are raised and wall-like, hard, irregular, and at some points undermined.

(*ii*) The floor of the ulcer is depressed and craterlike, fissured and nodular only near the edges.

(*iii*) The surrounding tissues are raised by tumor masses.

5. The boundaries of the tumor are not distinct and cannot be sharply defined.

By palpation:

1. The relation of the tumor to the skin covering it differs. In the upper half the skin may be raised in folds, while in the lower medial quadrant, adjacent to the ulcer and nipple, it is intimately attached to the nodular tumor. The retracted nipple cannot be drawn out.

2. The surface of the tumor is irregular and nodular.

3. The boundaries of the tumor are sharp and well defined upon the outer side only. The upper boundary is not sharply defined against the normal breast; below the tumor becomes fused with the deeper structures.

4. The tumor is situated in the left breast.

5. It is firmly attached to the underlying tissue, as it cannot be moved upon the thorax, and remains stationary when the pectoralis major contracts.

6. Its consistency is everywhere hard.

(B) The general examination reveals greatly enlarged, indurated lymph nodes in the left axillary and supraclavicular fossæ. There are no secondary nodules in the structures adjacent to the tumor. Nothing abnormal can be found in the viscera (especially the lungs and liver). The patient is anæmic and the general condition is bad.

(C) In making a diagnosis in this case it is important to remember that tumors in the part involved—the female breast—are very common. Benign growths, such as fibro-adenomas and lipomas, hypertrophy of the breast, and interstitial mastitis as well need not be considered in making a diagnosis in this case. The rapid growth, ulceration, and involvement of lymph nodes make the diagnosis of a malignant growth positive. The indurated lymph nodes, the hardness of the tumor, and the characteristic form of the ulcer speak for carcinoma, which is common in the breasts of women over forty years of age.

(D) The microscopic examination of a piece of tissue which sloughed off revealed a scirrhous carcinoma.

Diagnosis: Carcinoma of the left mammary gland.

CHAPTER V

GENERAL DISCUSSION OF THE TREATMENT OF TUMORS

Thorough operative removal of all parts of a tumor is the most certain therapeutic measure. It is usually indicated in the treatment of all tumors, excepting, of course, benign tumors giving rise to no trouble where the operation would be severe or mutilating and the cosmetic

results bad. Small tumors of the skin may be excised by an oval excision of the skin, and the resulting wound sutured. In the face, plastic operations are often required to close the defect following the removal of tumors of the skin, as immediate closure without such a procedure often leads to distortion of the parts and deformity.

The operative removal of malignant tumors is possible only when the diagnosis is made early and the growth is just beginning. Even then the excision must be carried into healthy tissues, especially in cases of carcinoma, and the neighboring lymph nodes must be exposed and removed even when there are no macroscopic changes.

A malignant tumor may be inoperable: (1) Because of the size of and extent of the primary growth; (2) because of metastases into the lymph nodes; (3) because of other metastases (disseminated, lymphogenous nodules in the surrounding skin and hæmatogenous metastases).

Inoperable Tumors.—For example, a carcinoma of the breast is inoperable if the tumor is firmly attached to the chest wall, or, even if the tumor is small, when the supraclavicular as well as the axillary lymph nodes are involved; when there are small, disseminated nodules in the surrounding skin, or metastases in the viscera (lung, liver) or in the bones (neck of the femur with spontaneous fracture, in the vertebra leading to pain, kyphosis, etc.).

Cauterization and its Indications.—Cauterization (with a thermocautery or caustics, in pedunculated fibromas of the mucous membrane with the loop of a galvano-cautery) is to be recommended for the removal of small, benign tumors, such as warts and pedunculated fibromas of the skin and mucous membranes only. It is to be discarded in the treatment of other forms of tumors, as the removal is not complete, and when there is a possibility of malignancy. Experience has shown that the irritation following cauterization hastens considerably the growth of malignant tumors. Besides, hideous, often deforming, scars remain after cauterization.

Ligation.—The ligation of the pedicle of pedunculated tumors (warts, pendulous fibroma) is performed by lay people and physicians who fear the knife. The object of ligation is to produce a necrosis and subsequent sloughing of the tumor. The great disadvantage of this procedure is that parts of the tumor, from which the growth recurs, often remain. The operations to be employed in the treatment of different varieties of tumors are described in their respective chapters.

Light Therapy: Indications and Contra-indications.—Light therapy is a modern method of treatment. The efforts which have been made to remove tumors by bloodless methods, and the discovery that the X-rays, radium, and Finsen rays produced inflammatory changes in the skin

often resulting in necrosis, led to a number of experiments dealing with the effect of the above-mentioned rays upon tumors. X-rays in particular seem to have some special action, as it has been demonstrated that warts, telangiectases, and carcinomas of the skin which have been exposed to the rays often disappear completely. This is apparently due to the degeneration of the tumor cells (von Mikulicz and Fittig, Pusey, Hyde, Bevan, Perthes, von Bruns).

In the treatment of superficial tumors the so-called soft (low vacuum) tube is employed. The diseased area is exposed, the tube being at a distance of 10 cm., from five to fifteen minutes, some days intervening between exposures, or from five to ten minutes for a number of successive days. The healthy surrounding skin should be protected by a lead plate while the treatment is being given. If the tumor is deeply situated a hard (high vacuum) tube should be employed, the rays from which penetrate more deeply. If the rays have the proper penetration the bones, when looked at through a fluoroscope, should cast a gray, not a black shadow. According to our present knowledge, the treatment should be continued only until an erythema of the normal skin, with or without vesicle formation, develops. Usually this occurs after two weeks. If the dosage is higher (harder tube or continued for a longer time) alterations in the vessels of the normal skin (degeneration of the intima and muscularis, Gassmann) which often lead to necrosis develop, and the rays are then no longer suited for the treatment of tumors (Perthes). Chronic "Roentgen-ray ulcers," which may occasionally become transformed into carcinomas, may develop after long and improper exposures.

In the treatment of benign tumors a trial with the X-rays is always permissible. In the treatment of malignant tumors the X-rays should be considered only for the small, flat carcinomas of the skin, especially those occurring upon the face, which pursue a chronic course and rarely form metastases. It should be kept in mind, however, that even in these cases, while there may be apparent healing, the growth may be extending more deeply beneath the scar, so that soon after superficial healing regressive changes may occur, leading to the formation of a deep ulcer which may even penetrate to bone. The simplicity, rapidity, and safety of the operative treatment are in marked contrast to the slowness and uncertainty of this method.

X-ray treatment of other operable malignant tumors, such as deep carcinomas and sarcomas, should be discarded. It can never replace the operative treatment, as the destruction and absorption are limited to the superficial parts of the tumor and occur but slowly during the course of weeks and months. In the treatment of malignant tumors, delay of complete removal merely favors the formation of metastases.

For example, of what value is it if a carcinoma of the lip heals after a number of exposures to the X-rays extending over a number of months, and in the meantime the regional lymph nodes have become so large and so firmly attached to the mandible and large vessels that the case is no longer operable? Such cases have been shown by specialists in skin diseases to demonstrate "the good results obtained by this new method of treatment." The dangers of metastases, which cannot be prevented by the X-rays, demand that this method be discarded in the treatment of malignant tumors which are still operable.

On the other hand the X-rays are of decided value in the treatment of inoperable, malignant tumors. Frequently such tumors have been seen to decrease in size, and even if the formation of metastases and the deeper extension of the growth are not prevented, the apparent improvement of the local condition is of inestimable value to the doomed patient.

In the treatment of inoperable tumors, it should be especially emphasized that the patient should not be deprived of the hope of recovery. The truth as to the nature of the disease should be concealed from the patient whenever it is possible. But when the surgeon is compelled, because of family relations or the demands of the patient, to depart from this rule, he should explain the nature of the lesion in a serious, earnest manner, and never treat it as a trivial, insignificant matter.

Local Treatment.—The objects of local treatment are the partial destruction of the tumor and healing of ulcerated areas. Partial destruction may follow the X-ray treatment, the parenchymatous injection of alcohol and zinc-chlorid solutions (twenty to fifty per cent). [These parenchymatous injections may produce a necrosis of parts of the tumor or a proliferation of the connective tissue, which as it contracts strangles, as it were, the epithelial cells.] Sometimes apparent improvement follows the internal administration of arsenic preparations and of the iodid of potassium. Ulcerated surfaces should be protected from infection (erysipelas, putrefactive phlegmon) by aseptic dressings and sterilization of the surrounding skin. The foul-smelling discharge may be lessened by using compresses of hydrogen peroxid, acetate of aluminum, etc. The actual cautery and caustics (moist compresses of from twenty to fifty per cent zinc chlorid according to Czerny) may be employed for the same purpose.

Treatment by Coley's Toxins.—Busch (1866), later von Bruns and Biedert, observed that occasionally a sarcoma underwent fatty degeneration, disappeared, and did not recur after an attack of erysipelas. Carcinomas may even become smaller, and Fehleisen reported a case in which a carcinoma subsided after an attack of erysipelas artificially produced. Many attempts have been made of late, especially by Coley,

to cure malignant growths by injections of sterile streptococcic cultures. [The mixed toxins of prodigiosus and streptococcus introduced by Coley have a distinct beneficial effect in sarcomas, and should be employed in all inoperable cases, as a small percentage are greatly benefited and some are even cured by this treatment.]

Morphin.—Morphin or codein should be given for the pain developing within and radiating from the tumor. In many cases it is the only drug which will render the life of the patient bearable.

If, as a result of erosion of one of the larger vessels, the hæmorrhage from the ulcerated mass is severe, all the methods at the command of the surgeon for control of hæmorrhage should be employed.

The general treatment should be symptomatic. Serum treatment up to the present time has given no results. Ehrlich's investigations, however, have demonstrated what lines should be followed in this work. By inoculation of mice with mouse carcinoma of low virulence, he has rendered them immune against mouse carcinoma of high virulence.

[An interesting experiment has been performed by Crile and Beebe, in which the blood of dogs with a rapidly growing sarcoma was replaced by direct transfusion with blood from sarcoma-immune dogs, resulting in the disappearance of the tumors.]

LITERATURE.—*Apolant.* Ueber den jetzigen Stand der Krebsforschung. Ther. d. Gegenwart, 1906, April.—*Borst.* Die Lehre von den Geschwülsten. Wiesbaden, 1902.—*v. Bruns.* Krebsbehandlung mit Röntgenstrahlen. Therap. d. Gegenwart, 1904.—*Cohnheim.* Vorlesungen über allgemeine Pathologie. Berlin, 1882.—*Czerny.* Warum dürfen wir die parisitäre Theorie für die bösartigen Geschwülste nicht aufgeben? Beitr. z. klin. Chir., Bd. 25, 1899, p. 243;—Ueber die Behandlung inoperabler Krebse. Chir. Kongr.-Verhandl., 1900, II, p. 1;—Ueber Heilversuche bei malignen Geschwülsten mit Erysipeltoxinen. Münch. med. Wochenschr., 1895, p. 833.—*Ehrlich.* Exper. Karzinomstudien an Mäusen. Zeitschr. f. ärtzl. Fortbild., 1906, p. 205, and Arbeiten aus dem K. Inst. f. experim. Therapie. Jena, 1906.—*Exner.* Ueber die bisherigen Dauerresultate nach Radiumbehandlung von Karzinomen. Deutsche Zeitschr. f. Chir., Bd. 75, 1904, p. 379.—*Fittig.* Ueber die Behandlung der Karzinome mit Röntgenstrahlen. Beitr. z. klin. Chir., Bd. 42, 1904, p. 505.—*Friedrich.* Heilversuche mit Bakteriengiften bei inoperablen bösartigen Neubildungen. Chir. Kongr.-Verhandl., 1895, II, p. 312.—*Gassmann.* Zur Histologie der Röntgenulcera. Fortschr. a. d. Geb. d. Röntgenstrahlen, Bd. 2, 1898–99, p. 199.—*Geissler.* Die Uebertragbarkeit der Karzinome. Arch. f. klin. Chir., Bd. 46, 1903, p. 655.—*Löwenthal.* Ueber die traumatische Entstehung der Geschwülste. Arch. f. klin. Chir., Bd. 49, 1895, p. 1.—*Lubarsch.* Zur Lehre von den Geschwülsten und Infektionskrankheiten. Wiesbaden, 1899.—*Luecke* and *Zahn.* Chirurgie der Geschwülste. Deutsche Chir., 1896.—*v. Mikulicz* and *Fittig.* Ueber einen mit Röntgenstrahlen erfolgreich behandelten Fall von Brustdrüsenkrebs. Beitr. z. klin. Chir., Bd. 37, 1903, p. 676.—*Perthes.* Ueber den Einfluss der Röntgenstrahlen auf epitheliale Gewebe, insbesondere auf das Karzinom. Chir. Kongr.-Verhandl., 1903, II, p. 525;—Versuche über den Einfluss der Röntgenstrahlen und Radiumstrahlen auf die Zellteilung. Deutsche med. Wochenschr., 1904, p. 632;—Zur Frage der Röntgentherapie des Karzinoms. Arch. f. klin. Chir., Bd. 74, 1904, p. 400.—*Petersen* and *Exner.* Ueber Hefepilze und Geschwülstbildung. Beitr.

z. klin. Chir., Bd. 25, 1899, p. 768.—*Répin.* La toxithérapie des tumeurs malignes. Revue de chir., 1895, vol. 15.—*Ribbert.* Geschwülstlehre. Bonn, 1904;—Beitr. z. Entstehung d. Geschwülste. Bonn, 1906.—*Schmieden.* Erfolgreiche experimentelle Verlagerung von Nebennierengewebe. Deutsche Zeitschr. f. Chir., Bd. 70, 1904, p. 453. —*Virchow.* Die krankhaften Geschwülste. Berlin, 1863.—*Vischer.* Ueber Sarkomübertragungsversuche. Beitr. z. klin. Chir., Bd. 42, 1904, p. 617.—*Wilms.* Implantation und Wachstum embryonaler Gewebe. Chir. Kongr.-Verhandl., 1904, I, p. 287.—*Wyss.* Zur Entstehung d. Röntgenkarzinomes der Haut u. s. w. Beitr. zur klin. Chir., Bd. 49, 1906, p. 185.—*Ziegler.* Lehrbuch der allgemeinen Pathologie. Jena.

II. DIFFERENT VARIETIES OF TUMORS

A. CONNECTIVE-TISSUE TUMORS

CHAPTER I

FIBROMAS

A BENIGN tumor composed of fibrous tissue is called a fibroma. Fibromas occur as round or pedunculated tumors upon the skin and mucous membrane, often becoming very large. They may be congenital or develop at any time of life.

Mode of Growth.—They grow slowly in an expansive way, and therefore belong to the benign tumors. The surrounding tissues are displaced or undergo a pressure atrophy. Pressure atrophy may occur even when the adjacent structure is bone. There is no tendency to recur after excision except in that variety known as keloids.

Histological Characteristics.—A fibroma is composed of cells, connective-tissue fibrillæ and blood vessels. The first, with their long, oval nuclei and narrow zone of cytoplasm, resemble the cells of ordinary fibrous tissue. Cells with large nuclei and rich in protoplasm also occur in rapidly growing fibromas. The intercellular substance is composed of a large number of fibrillæ. Sometimes these fibrillæ are single, at other times grouped to form a bundle of fibrillæ which are closely or loosely arranged. Fibromas present a number of different forms, depending upon the number of cells and the grouping of the fibrillæ.

The Hard Fibroma, or Desmoid, and Soft Fibroma.—The hard fibroma, or desmoid, white and shining upon section, is composed of thick, coarse bundles of fibers with few cells. The soft fibroma (fibroma molluscum) is grayish white upon section, and is composed of delicate bundles of fibrillæ or single fibers, between which are tissue fluids containing lymphocytes and leucocytes, often giving to the tumor an œdematous appearance.

Relation to Surrounding Structures.—Fibromas are usually intimately connected with the surrounding tissues, but in spite of this they may easily enucleate in many cases. If the tumor develops in muscles it will contain muscle fibers. Nerve fibers and glands are found in fibro-

mas developing in nerves and glandular organs. Bone is not infrequently found in periosteal fibromas. As these fibromas develop from the fibrous tissue of the periosteum, the bone which they contain is of periosteal origin.

Many fibromas are the result of disturbances of development; for example, the congenital tumors of the skin and nerves and those covering an encephalocele or a spina bifida occulta. In many forms (keloid, elephantiasis nervorum) there is a distinct, even hereditary, predisposition. Some fibromas develop from inflammatory proliferations.

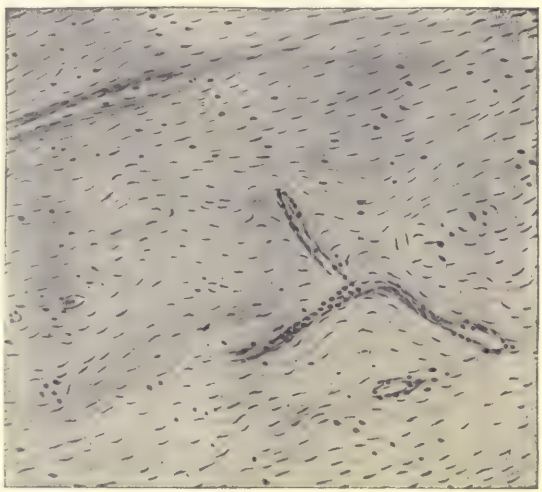


Fig. 283.—Hard Fibroma with Few Vessels.

It is not possible to make a sharp distinction between fibromas and the two other forms of tissue proliferation. *Chronic inflammatory growths* of the skin and subcutaneous tissue and *congenital hypertrophies* (partial giant growth) lead to the formation of tumorlike, elephantous masses. If these are sharply circumscribed and distinguished from the surrounding structures by a more independent growth—for example, if they contain lobulated or pedunculated parts—they are to be regarded as lobulated elephantiasis and classified with fibromas. It is often difficult to differentiate between a fibroma and a fibrosarcoma, which may develop from the former. If the tumor is rich in cells with large, oval nuclei undergoing rapid division and with a large amount of cytoplasm, and there is but little intercellular substance, the tumor should be regarded as a fibrosarcoma.

The vascularity of fibromas varies greatly. If the blood vessels, which are represented by spaces of different size lined by endothelium, are present in large numbers (e. g., in many nasal and pharyngeal polypi) the tumor may be designated as a *fibroma teleangiectaticum*, or, if it contains large sinuses, as a *fibroma cavernosum*. The *fibroma lymphangiectaticum* is provided with numerous dilated lymphatic vessels.

Regressive Changes.—A number of regressive changes may occur in fibromas. The vessels may be occluded by the pressure of neighboring

structures or as the result of torsion of the pedicle, and the tumor may then undergo necrosis. As the result of liquefaction of the intercellular substance, areas containing mucoid tissue (*fibroma myxomatodes*) and cavities (*fibroma cysticum*) develop.

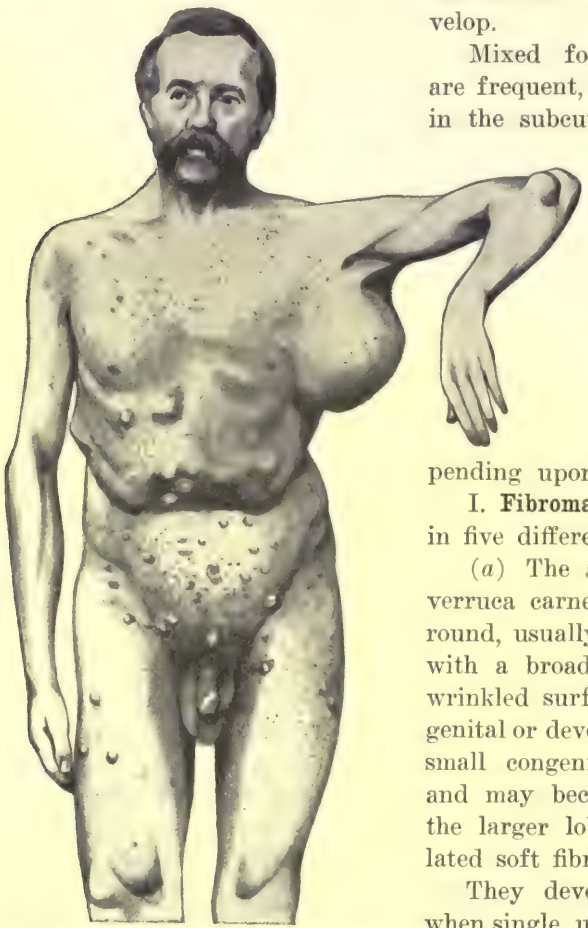


Fig. 284.—Fibromata Mollusca of the Skin and Sarcoma of the Left Axillary Fossa.

Mixed forms of these tumors are frequent, fibrolipomas occurring in the subcutaneous and subserous tissues, fibromyomas in the uterus.

Fibromas are common, occurring upon different parts of the body, some parts being more frequently involved than others. Eight varieties may be differentiated, depending upon the tissues involved.

I. **Fibromas of the skin** appear in five different forms:

(*a*) The *soft wart* (flesh wart, verruca carnea) occurs as a small, round, usually pigmented formation with a broad base and smooth or wrinkled surface. It is either congenital or develops in childhood from small congenital pigmented moles, and may become transformed into the larger lobulated and pedunculated soft fibroma.

They develop most frequently, when single, upon the face and neck. If associated with a general fibromatosis of the nerves, they may be disseminated over the entire surface of the body, alternating with numerous flat, wartlike nævi and larger fibromas.

According to Soldan, histologically they are soft fibromas rich in cells, and develop from the connective-tissue cells of the cutaneous nerves.

They can be easily differentiated from ordinary warts, which are so frequent upon the hands of children. The ordinary warts differ from

those described above: (1) In that they are transferable to different parts of the same individual, as the experiments of Jadassohn and Lanz have shown; (2) they are the result of the hypertrophy of the papillæ and the skin covering them and depending upon the condition of the latter; their surfaces are smooth, rough, or fissured. There are some other forms of congenital warts or papillomas which belong to the fibro-epithelial tumors.

(*b*) The *multiple, painless tumors of the skin*, which are sometimes nodular, sometimes pedunculated, are designated as *fibromata mollusca*. They vary in size, and, according to von Recklinghausen, develop from the cutaneous nerves, belonging therefore to the neurofibromas. Not infrequently they contain the most delicate plexiform neuromas (*vide* p. 793).

FIG. 285.—LOBULATED ELEPHANTIASIS (ELEPHANTIASIS NERVORUM).

(*c*) Under the term *lobulated elephantiasis* are grouped a number of different fibrous growths of the skin which resemble each other in that they are composed of long, pendulous growths, folds, flaps, or

masses. Part of these growths belong to the lymphangiomas and hæmangiomas (elephantiasis lymph- and hæmangiectatica); part are soft fibromas which, like the nodular soft fibromas of the skin, develop from the connective tissue of cutaneous nerves, but they also contain numerous lymphatic and blood vessels (von Esmarch and Kulenkampff).

The nerve form of elephantiasis may be congenital or develop in early childhood from soft fibromas. The tumors occur most frequently upon the face and scalp, upon the neck, and about the region of the shoulder. They are covered by thin, wrinkled skin which is often pigmented and covered with hair.

There are frequently found associated with the fibrous growths occurring in this form of elephantiasis, other tumors which belong to the neurofibromas, such as the soft wart, fibromata mollusca, the plexiform neuromas in the base of the lobulated growths, and finally fibromas of the larger nerves; changes which von Bruns has placed in one group and to which he has given the name of *elephantiasis nervorum*. Almost all of these forms are associated with congenital changes in the skin, indicated by small and large, usually flat, light-brown pigmented areas. Just as a soft wart may become transformed into a larger fibroma and these into lobulated formations, so, according to Soldan, each pigmented mole at any time may become transformed into a soft wart or into multiple fibromas of the skin. The cell columns of these pigmented nævi with alveolar arrangement are, according to the researches of this investigator, neither of epithelial nor of endothelial origin, but are derived from the connective tissues of the nerves of the cutis. These histological findings correspond perfectly to the clinical picture as the flat, more rarely, verrucous nævi, soft warts, fibromas of the skin, and deeper nerves are frequently associated.

FIG. 286.—FIBROMA PENDULUM.

Independent growths developing from inflammatory hyperplasias,

which are secondary to repeated attacks of erysipelas, and infections with filaris sanguinis hominis, do not belong to lobulated elephantiasis.

(*d*) The *hard fibroma of the skin* is less frequently of congenital origin than is the soft fibroma. It appears as a small, hard nodule or as a slowly growing fungoid, pendulous tumor with a long, thin pedicle.

FIG. 287.—FUNGIFORM, HARD FIBROMA OF THE SKIN WITH SECTION OF THE SAME.

The latter form may develop at any period of life. It occurs most frequently upon the back, the inner side of the arms and thighs. There are also transitional forms to the soft fibroma. Upon section the tumor is sharply defined against the subcutaneous fat, and its surface is covered by a thin layer of subepithelial connective tissue.

Diagnosis.—The diagnosis of fibromas of the skin is not difficult, notwithstanding the great number of different forms that are met with. If it is found upon section or microscopic examination that the epi-

thelium has proliferated to form part of the tumor, it should be placed in the fibro-epithelial group (Ribbert). Growths occurring in this form of elephantiasis, which become smaller under pressure, are either closely related to or belong to lymphangiomas and hæmangiomas.

Fig. 288.—Fibroma of the Skin with Broad Base as It Appears upon Section.

Treatment. — These growths should be excised when they become so large or are so situated that they cause trouble. If the growths in elephantiasis are extensive, partial excision at a number of different sittings is indicated. In the congenital forms the proliferated connective tissues and the subcutaneous nerves must be carefully removed in order to prevent recurrence. The defects resulting from operations upon both the congenital and acquired forms of elephantiasis should be repaired by skin grafting or plastic operations.

(*c*) Keloids form a special group of fibromas of the skin. They are hard tumors, relatively rich in cells, and are composed of thick bundles of connective-tissue fibrils which frequently become transformed into homogeneous, collagenic trabeculæ. Sometimes they occur as painless, red, indurated thickenings of the skin, sometimes as nodular new growths of considerable size, and sometimes in the forms of tumors sending outgrowths into the adjacent healthy tissues.

The growth involves only the reticular layer of the cutis. It never extends to the deeper structures (Fig. 289). A keloid is therefore always displaceable with the skin, and may be raised from the underlying structures. The surface of a keloid is red and shining and is covered by a layer of epidermis which contains no papillæ and is not cornified, and by a thin layer of vascular connective tissues. The tissues composing a keloid gradually fuse with the healthy surrounding tissues, and the boundaries of such a growth are therefore never sharp and distinct. The neighboring tissues are pushed aside and separated by the expansive growth of these tumors. They contain neither elastic fibers, hair, nor sebaceous glands.

Cicatricial and Spontaneous Keloids.—The majority of keloids develop within a cicatrix. They increase gradually in size, often for a number of months, and then remain stationary or in rare cases disappear spontaneously. They develop most commonly in scars resulting

from burns of the third degree and from cauterization, but also in suppurating wounds and wounds healing by primary union, from chronic ulcers, vaccination scars, and from contusions of the skin. Since Alibert's description (1814) the keloids following wounds have been spoken

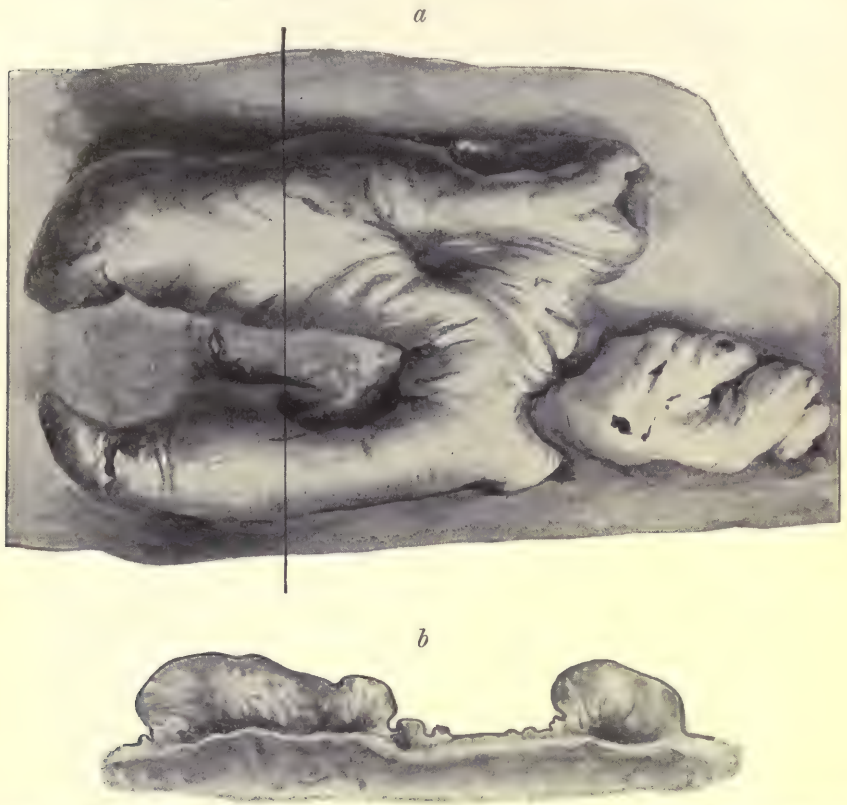


FIG. 289.—*a*, KELOID WHICH DEVELOPED UPON THE FOREARM OF A CHILD EIGHT YEARS OF AGE AFTER A SCALD. *b*, SECTION THROUGH THE SAME.

of as cicatricial keloids and differentiated from spontaneous keloids. Histologically they are alike, and the spontaneous development of the latter is probably only apparent, as they develop after some insignificant wound or injury of the cutis in which there has been no separation of the epidermis.

The Hypertrophied Scar and Differences Between It and a Keloid.—The hypertrophied scar, which appears as red, frequently very painful, hard, flat, or irregular tumorlike growth at the site of a former injury, is not to be regarded as a keloid. It is not strictly speaking a new growth, but is the result of excessive scar formation in the most

superficial layers of the cutis. A hypertrophied scar develops most frequently from infected wounds. Wavy, loose, connective-tissue bundles without deposits of collagen (Goldmann), normal connective-tissue cells, frequently also cellular infiltrations and isolated hair follicles are

FIG. 290.—KELOID DEVELOPING IN A LAPAROTOMY WOUND (YOUNG WOMAN, TWENTY YEARS OLD).

found in a hypertrophied scar, and these histological findings enable one to differentiate between such a scar and a keloid.

A hypertrophied scar also differs from a keloid in that the scar tissue slowly undergoes the normal changes and eventually almost, if not completely, disappears.

Positions in which Keloids are Most Common.—The position of the keloid is naturally determined by the site of the injury. There are, however, parts of the body in which keloids are especially prone to develop—for example, they follow more frequently injuries to the lobe

of the ear, breast, face, and vaccination scars of the upper arm, than they do injuries of the skin of the palm of the hand and the sole of the foot. Keloids are most frequent in people of middle age.

Recurrence after Extirpation.—The great tendency to recur is the most important characteristic of keloids. Even after complete extirpation a new keloid may develop within some weeks within the resulting cicatrix. It makes no difference whether the wound has been sutured,

Fig. 291.—A Keloid which Developed in a Sutured Wound of the Arm after the Excision of a Keloid which Developed in a Vaccination Scar.

closed by a plastic operation, covered by skin grafts, or has healed by secondary intention. Small nodules will even develop from the stitch holes of a sutured wound (Figs. 290 and 291). Only in rare cases is

there no recurrence after extirpation. Von Bergmann observed a case in which there was no recurrence after seventeen years. In spite of the fact that they recur, keloids do not belong to the malignant tumors, as they do not form metastases.

Multiple Keloids.—The development of multiple growths is another important characteristic of keloids. A person with a keloid may develop other similar growths at the site of any injury, provided the injury has involved the cutis (Lauenstein). There are cases, however, in which extensive injuries and operation-wounds of parts of the body some distance from the site of a keloid heal normally. The different results following wounds indicate that there is a predisposition to keloid formation which in one case may be general, in another case local. The fact that heredity is a factor in some cases also speaks for a special predisposition.

FIG. 292.—RECURRENCE AFTER EXCISION OF A SPONTANEOUS KELOID.

Causes of Keloid Formation.—Negroes are peculiarly liable to develop keloids. Nothing is known of the essential cause of keloid formation. The theory advanced by Goldmann that absence of the connective-tissue bundles of the cutis, resulting from injury, is the cause, is not a satisfactory explanation.

Diagnosis.—The diagnosis of keloids is not difficult. They may be confused with large, irregular, hypertrophied scars (e. g., after burns and cauterization).

Treatment.—The great tendency to recur must always be considered before treatment is instituted. Excision is not to be advised, especially if recurrences have already developed. Goldmann believes that recurrence may be prevented if the wound following excision of a keloid is immediately covered by large epidermal grafts. According to the experience of the author proliferation of the granulation tissue is prevented by the firmly agglutinating epithelium. Recurrences cannot be

prevented with certainty either by this procedure, suturing of the wound, or by plastic operations.

A number of different chemical agents have been used in the treatment of keloids. Thiosinamin, first introduced by Hebra in 1892, is the best agent. It is frequently used in the form of a fifteen per cent alcoholic solution or as a ten per cent aqueous glycerin solution, as recommended by Duclaux. The solution should be injected directly into the keloid. After a number of injections of 1 c.c. the keloid becomes smaller. If the keloids or hypertrophied scars are very large the injections must be continued for a number of months. The injections are quite painful to a number of patients. This method, which at least causes a reduction in the size of the keloid, is to be preferred to excision. Fibrolysin (Merck) soluble in water is a double salt of thiosinamin and sodium salicylate. [Hyde and Ormsby have removed a number of keloids with the X-ray. This should be to-day the treatment of choice.]

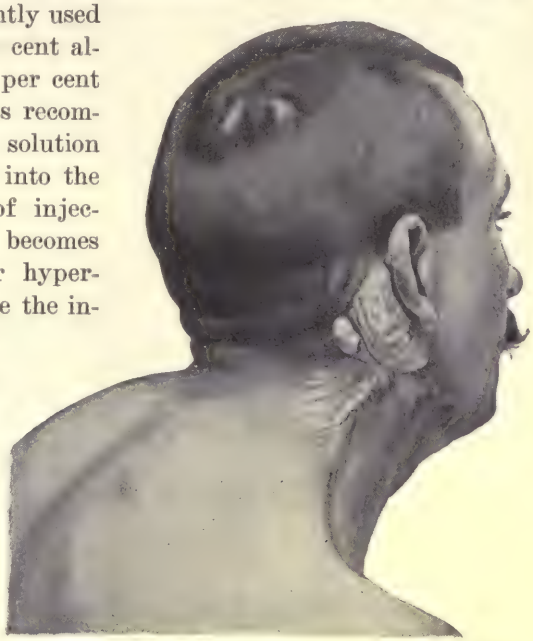


FIG. 293.—ANOTHER RECURRENCE AFTER FOUR YEARS.

II. **Fibromas of the subcutaneous tissues** are much less common than those of the skin. They may occur upon almost any part of the body at any period of life, and growing slowly may attain considerable size. The skin covering them is of normal appearance, and may be raised from the tumor. These tumors are encapsulated and may be displaced upon the underlying fascia. They first give rise to symptoms by pressure upon nerves.

The *diagnosis* is based upon the slow growth, hard consistency, and encapsulation. Extirpation is not difficult.

III. **Fibromas of the mucous membranes** occur most frequently in the nose. They are usually multiple. The pedunculated or lobulated nasal polypi are composed of loose or firm fibrous tissue which contains large vessels, and are covered by a stratified epithelium and a thin layer of submucous tissue. It is often difficult to distinguish between them and polypoid, inflammatory growths of mucous membranes.

Similar but smaller multiple tumors are found in the larynx; more rarely in the gastro-intestinal canal, the urethra, and bile passages. Small, circumscribed, smooth fibromas may also be found in the mouth cavity (on the tongue, in the floor of the mouth, and upon the gums).

Laryngeal and nasal polypi should be grasped with special forceps and torn away. Sometimes it is necessary to split the nose or larynx in order to remove these tumors.

IV. **Fibromas of the fasciæ and aponeuroses** are hard, nodular, painless growths. They may be single or multiple. These tumors develop most frequently in the abdominal wall (desmoid of the abdominal wall), taking their origin most often from the posterior sheath of the rectus muscle, more rarely from the anterior sheath, from the aponeuroses of the oblique muscles, from the transversalis fascia and the linea alba. They grow slowly, becoming as large as or larger than a fist, and separate the adjacent muscles. Their growth is hastened by pregnancy. The muscle from the fascia of which the fibroma develops undergoes in places a pressure atrophy as the tumor extends along the intramuscular connective-tissue bundles (Fig. 294).

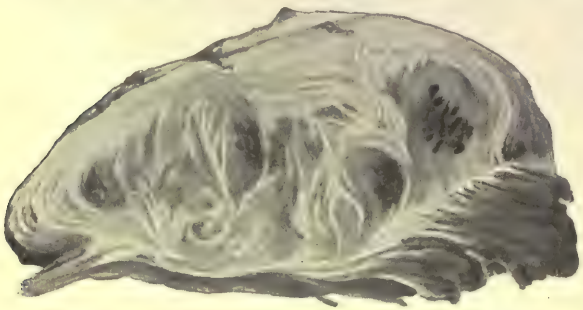


Fig. 294.—Fibroma of the Internal Oblique Muscle of the Abdomen in a Female Patient Twenty Years of Age (Natural Size).

In isolated cases a number of these tumors have been found in the abdominal wall, one developing after another. Recurrence may occur after excision, but it is rare (*vide* Pfeiffer).

These tumors, as a rule, develop only in women who have borne children, and it is probable that trauma (slight laceration of the aponeurosis) is the etiological factor in their development.

The diagnosis is based upon the position of the tumor in the abdominal wall, its hardness, slow growth, and round form. It is important to determine the relation of the tumor to the muscles by palpation and by rendering the muscles tense.

After removal of the tumor, the defect in the muscles should be sutured in order to prevent a hernia.

Fibromas of the neck form another clinical group of tumors arising from fasciæ and aponeuroses. They develop in the posterior part of the neck from the cervical aponeurosis, in the anterior part from the sheath of the vessels, at the sides from the intermuscular connective

tissues. They may take their origin from the periosteum of the vertebræ and from the dura mater of the cervical part of the spinal cord (de Quervain).

V. **Fibromas of the periosteum** are most common upon the maxilla and mandible and at the base of the skull. They are found almost always in young people and very rarely develop on other bones. They belong to the hard form of fibromas, and often contain many large vessels, frequently cavernous tissue. Occasionally they contain bone, as they develop from periosteum.

Epulides.—Fibromas of the jaw, together with different forms of sarcomas, form part of the clinical group of tumors known as epulides (epulis, from ἐπί, meaning upon, and οὖλις, meaning gum), therefore those tumors which are situated upon the gums. Developing from the periosteum of the alveolar processes, they grow between the teeth as small nodules covered with mucous membrane. They give rise to symptoms only when they have attained considerable size or ulcerate and

Fig. 295.—Fibrous Nasopharyngeal Polyp which has Invaded the Antrum of Highmore.

bleed. Lobulated and round forms of periosteal fibromas occur upon other parts of the jaw, especially upon the upper. Central fibromas are most common in the jaw bones, developing from the connective tissue of the bone marrow, from the blood vessels or nerves, perhaps also from displaced tooth-buds (Blauel). The bone gradually undergoes a

pressure atrophy and becomes expanded as these tumors grow. Finally they may rupture through the thin shell of bone covering them. A tumor of the upper jaw may then grow into the antrum of Highmore.

Fibromas of the Vault of Pharynx.—The hard fibromas which are found in the vault of the pharynx and develop from the periosteum of the basilar part of the occipital and adjacent bones are known as fibrous nasopharyngeal polypi. They are covered by the epithelium of the epipharynx and grow in the directions in which there is the least resistance. These tumors are most common in the male sex between the fifteenth and twenty-fifth years. First they occlude the posterior nares. After they have filled the nasopharyngeal space, they extend into the pterygopalatine fossa. The surrounding bone undergoes a pressure atrophy, so that finally tumor masses invade the accessory sinuses of the nose, the sphenoidal sinus, and the skull cavity (Figs. 295 and 296). If the tumor ulcerates, severe hæmorrhages from the fairly large vessels may occur.

FIG. 296.—THE SAME TUMOR SHOWING INVASION OF THE SKULL CAVITY.

These tumors are not infrequently mistaken for malignant tumors which develop from the base of the skull or upper jaw and extend in much the same way.

It is often necessary to perform a preliminary operation (temporary resection of the upper jaw) in order to render these tumors accessible, so that a radical removal may be performed and recurrences prevented.

VI. **Of the glandular organs,** the breast is the most frequent seat of fibromas. Fibromas occurring in this organ frequently contain glandular elements (*vide* Adenoma). Part of the fibromas, fibroadenomas, and fibrolipomas occurring in the kidney are the result of developmental disturbances. These tumors are most frequently situated in the pyramids, enclosing renal tubules, at the hilus, and beneath the capsule, and may attain considerable size.

VII. **Fibromas of the nerves** (fibromata nervorum), or less correctly speaking, neurofibromas, are fairly common tumors. They develop from the connective tissue, the ends and perineurium of nerves, and are traversed by nerve fibers which have been separated by the growths but

have not proliferated. Usually these tumors are multiple, often being present in large numbers.

They develop most frequently upon the delicate cutaneous nerves. Their relation to soft fibromas of the skin and elephantiasis nervorum has already been discussed (*vide* Fibromas of the Skin, p. 781).

Fibromas developing upon the larger nerves and nerve trunks, including the sympathetic nerves and the roots of cranial and spinal nerves, lead to the formation of flasklike or spindle-shaped swellings; sometimes to the formation of large tumors or nodular thickenings which may be distributed over a considerable extent of the nerves involved.

The plexiform neuroma is a nodular, fibrous degeneration of a particular nerve, usually of one of the subcutaneous nerves. As it develops, the nerve involved becomes transformed into a thickened, nodular, wreathlike, twisted, tortuous, circumscribed mass. Plexiform neuromas may also develop in the terminal filaments of cutaneous nerves, and often lie concealed beneath soft fibromas of the skin and lobulated growths occurring in elephantiasis.

The plexiform neuroma, if not present at birth, begins to develop in early childhood. Tumors of the larger nerves grow slowly and appear in middle age. Even these larger tumors have some relation to congenital changes in the nerves, for they are often apparently merely a late manifestation of those changes which are comprised under the term elephantiasis nervorum.

Fig. 297.—Nerves Dissected Free from a Subcutaneous Plexiform Neuroma Removed from the Occipital Region of a Child Four Years Old.

The cause of the formation of fibromas of the nerves, likewise of all the forms of elephantiasis of nerves, must be sought in some disturbance of normal development as a result of which, perhaps, there is an irregular distribution and arrangement of the connective-tissue elements of the nerves permitting of independent growth (Ribbert).

The following facts may be cited to support this theory: (1) These tumors develop in early childhood; (2) they are multiple; (3) an heredi-

tary history may be elicited or a congenital predisposition shown as different forms of elephantiasis nervorum (either neurofibromas, soft fibromas of the skin, soft warts or pigmented nævi) may be demonstrated in different members of the same family.

The clinical significance of fibromas of nerves varies. The fibroma molluscum, and even fibromas of larger nerves, may give rise to no symptoms. The most frequent symptom is pain, which radiates along the nerve from the tumor toward the periphery. It is increased by movements and pressure. However, slight functional disturbances, very rarely sensory and motor paralyses, may develop. Frequently these tumors compress adjacent nerves or parts of the brain and spinal cord (fibromas at the point of origin of cranial and spinal nerves).

Isolated fibromas of nerves do not recur after extirpation. Sometimes there is a tendency to progressive development of tumors upon all the nerves of the body (von Büngner). In twelve per cent of the cases of general fibromastosis of the nerves, sarcomas (fibrosarcoma and myxosarcoma) develop from one of the nodules, which, according to Garrè, should be called secondary malignant neuromas to distinguish them from the primary sarcomas developing in nerves.

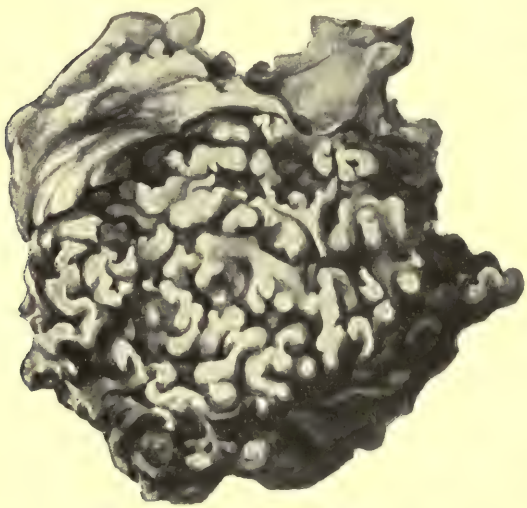


Fig. 298.—Plexiform Neuroma of the Subcutaneous Nerves of the Thorax in a Boy Eight Years of Age. (The tumor, about as large as a small plate, was flat and covered by normal skin, which, however, was somewhat adherent to it. Surface of tumor somewhat nodular. Pain upon pressure. Tumor well encapsulated and not attached to underlying structures. Many flat, pigmented moles in the skin adjacent to the tumor.)

The *diagnosis* of fibromas of nerve is not difficult when they are multiple and the larger nerves are involved. Where there are isolated nodules situated upon deep nerves, one is often in doubt whether the enlargement is a lipoma, a lymph gland, etc., which is pressing upon an adjacent nerve and giving rise to a radiating pain. Other changes, such as light-brown, flat, pigmented areas, which may be localized or distributed over the entire surface of the body, are frequently associated with these fibromas and are of considerable importance as a diagnostic aid.

Treatment.—The treatment consists of resection of the part of the nerve involved, when the growth is limited enough to render this possible.

In plexiform neuromas the skin covering them, which has undergone an elephantiasislike hyperplasia, should also be removed. Recurrences develop from thickened nerves which are left behind.

Fig. 299.—Large Fibroma (25 cm. in Diameter) Removed from the Mesentery of a Male Patient Forty-one Years of Age. During the operation it was necessary to resect about two feet of the small intestine which were adherent to the tumor. (Lexer.)

VIII. **Fibromas of the peritoneum** develop from the subserous tissue of the mesentery, mesocolon, and omentum, and from the retroperitoneal tissues. They grow slowly, forming hard, somewhat nodular tumors, and produce different symptoms depending upon their relation to the viscera.

These tumors often contract firm adhesions with the intestines or become so closely related to the blood vessels of the intestines that it is often necessary to resect some length of the intestinal loops in removing them.

Literature.—*Adrain.* Ueber Neurofibromatose und ihre Komplikation. Beitr. z. klin. Chir., Bd. 31, 1901, p. 1.—*v. Bergmann.* Demonstration eines vor 17 Jahren wegen eines grossen Keloides operierten Patienten. Verhandl. d. Berl. med. Gesellsch., 1903, I, p. 206.—*P. Bruns.* Das Rankenneurom. Beitr. z. klin. Chir., Bd. 8, 1892, p. 1.—*v. Büngner.* Ueber allgem. mult. Neurofibrome des periph. Nervensystems und Sympathikus. Chir. Kongr.-Verhandl., 1897, II, p. 298.—*Courvoisier.* Die Neurome. Eine klin. Monographie. Basel, 1886.—*v. Esmarch* and *Kulenkampff.* Die elephantiastischen Formen. Hamburg, 1885.—*Garrè.* Ueber sek. malign. Neurome. Beitr. z. klin. Chir., Bd. 9, 1892, p. 465.—*Kümmel.* Nasenrachenfibrome. Im Handb. d. prakt. Chir., Bd. 1, 2. Aufl.—*Lanz.* Leontiasis mit generalisiertem Fibr. moll. Deutsche Zeitschr. f. Chir., Bd. 58, 1901, p. 580;—Experim. Beiträge zur Geschwulstlehre. Deutsche med. Wochenschr., 1899, p. 313.—*Ledderhose.* Die chir. Erkrankungen der Bauchdecken. Deutsche Chir., 1890.—*Lewandowski.* Ueber Thiosinamin und seine Anwendung. Therap. d. Gegenwart, 1903.—*Lexer.* Operation eines Mesenterialfibromes mit ausgedehnter Resektion des Dünndarmes. Berlin klin. Wochenschr., 1900, No. 1.—*Olshausen.* Ueber Bauchwandtumeren, spez. üb. Desmoide. Zeitschr. f. Geburtsh. u. Gynäk., Bd. 41, 1899, p. 271.—*Pfeiffer* Die Desmoide der Bauchdecken

u. ihre Prognose. Beitr. z. klin. Chir., Bd. 44, 1904, p. 334.—*de Quervain.* Ueber die Fibrome des Halses. Arch. f. klin. Chir., Bd. 58, 1899, p. 1.—*v. Recklinghausen.* Ueber die multiplen Fibrome der Haut und ihre Beziehung zu der mult. Neuromen. Berlin, 1882.—*Soldan.* Ueber die Beziehungen der Pigmentmäler zur Neurofibromatose. Arch. f. klin. Chir., Bd. 59, p. 261, 1899.—*Wilms.* Zur Pathogenese des Keloid. Beitr. z. klin. Chir., Bd. 23, 1899, p. 149.

CHAPTER II

LIPOMAS

LIPOMAS are tumors in which is reproduced the structure of normal fatty tissue, and they are therefore composed of more or less lobulated, yellowish masses. They may be single or multiple, and in the latter case they are often symmetrically placed. Lipomas are decidedly benign and do not recur after complete extirpation. They are rarely congenital, developing most frequently in individuals from thirty to fifty years of age and in the female sex.

Macroscopic and Microscopic Appearance.—Lipomas, excepting the small, multiple, symmetrical forms which sometimes develop within a few months, grow very slowly. They develop from large cells which in earlier life are fat free, and become transformed into fat cells by the deposition and coalescence of fat droplets within their cytoplasm. Usually the cells found in a lipoma are larger than those occurring in normal fatty tissue. Groups of these fat cells, held together by a capillary network, form a small fat lobule which, using a favorite comparison, bears the same relation to its nutrient artery that grapes do to the lateral branches of the stem. The lobules, however, are not separate and distinct as in normal fatty tissue, but are united by connective-tissue trabeculæ into large lobes and fingerlike processes. The surface of the tumor is always provided with a thin connective-tissue capsule, sending trabeculæ into deep furrows between the different lobes, which are often the size of a hen's egg. Even when non-lobulated lipomas, which are rare, are cross-sectioned, thin connective-tissue trabeculæ may be seen passing from the capsule into the depths of the tumor.

The capsule of a lipoma is usually but loosely attached to the surrounding tissue. If, however, the tumor is exposed to irritation (rubbing of the clothing, pressure during work, etc.), the capsule becomes thickened and contracts adhesions with the skin and underlying tissues. The capsule then becomes fused with the subcutaneous fat and extirpation is rendered difficult.

Independent of General Nutrition.—A lipoma does not decrease in size when a patient emaciates (Virchow). The complete independence of the growth is best shown by this fact.

Blood Supply.—The few and relatively small vessels supplying a lipoma develop from the subjacent tissues and pass into the tumor with the interlobular connective tissue. Frequently but two or three arteries are found even in the large lipomas.

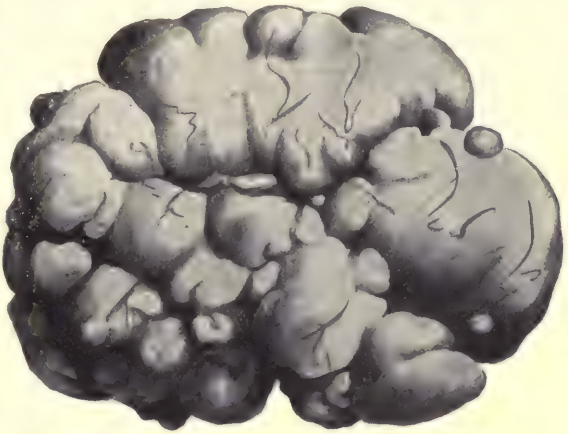


FIG. 300.—EXTIRPATED SUBCUTANEOUS AND INTERMUSCULAR LIPOMA.

Method of Growth.—The surrounding tissues are displaced by the tumor as it grows, and large processes are sent out into the loose, yielding interspaces of the surrounding structures. In this way a lipoma grows between muscles and tendons or along large blood vessels, extends from the palm to the dorsum of the hand between the metacarpal bone, or, developing from the subserous fat, forces its way

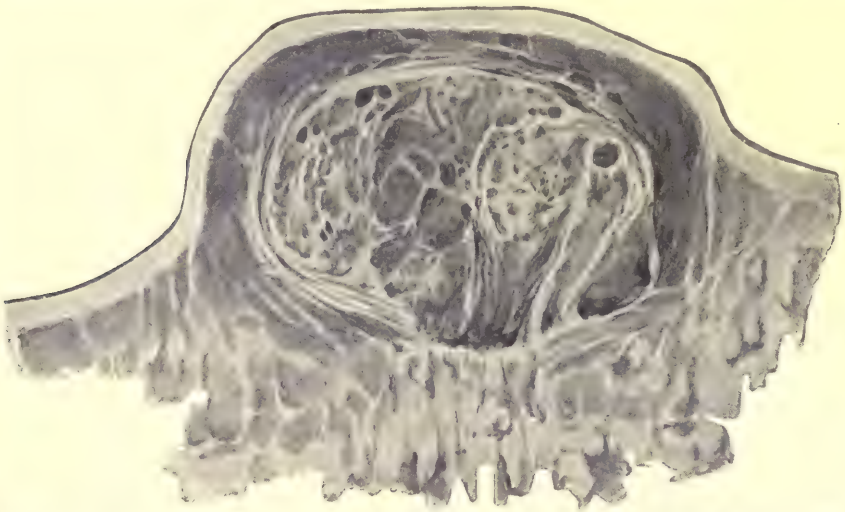


FIG. 301.—SECTION OF A SUBCUTANEOUS FIBROLIPOMA OF THE GLUTEAL REGION. The skin and a thin layer of subcutaneous fat pass over the surface of the encapsulated tumor.

through the femoral and inguinal canals or the linea alba and prepares the way for hernia.

A lipoma, which is usually soft, may become hard if the fibrous tissue is increased in amount (soft and hard lipomas). Depending upon whether the fatty or fibrous tissue predominates, one speaks of a *lipofibroma* or a *fibrolipoma.* If there is a marked development of blood vessels (proliferation and dilatation), the tumor is spoken of as an *angiolipoma.* If the fibrous tissue has become transformed into mucoid tissue, or if there are smooth muscle fibers in the tumor, it is described as a *myxolipoma* or *myolipoma.*

Regressive Changes.—Calcification, occasionally ossification of the septa, œdematous changes and liquefaction resulting in the formation of oillike masses (oil cysts) occur, especially in the larger tumors. Nutritional disturbances resulting in necrosis of part of the tumor and the skin covering it, with subsequent erosion of vessels and putrefactive changes may also develop in the larger growths.

Fig. 302.—Subcutaneous Lipoma of the Arm.

Origin and Causes of Lipoma.—Many lipomas, especially those occurring as heteroplastic tumors in viscera and tissue normally containing no fat, and as congenital tumors situated over defects in the skull and vertebræ (encephalocele, spina bifida occulta), develop from displaced germinal tissues. A hereditary influence has been observed in rare cases only (Blaschko). Grosch, Köttnitz, Payr, and others have suggested that the multiple, symmetrical lipomas are of trophoneurotic origin, and the first has attempted to show that certain tumors are prone to develop upon certain parts of the body because of anatomical conditions and structural peculiarities. After an exhaustive study concerning the dis-

tribution of lipomas upon the surface of the body, Grosch came to the conclusion that lipomas are most common in those areas where there are the fewest glands, and least common where the glands are most numerous, that therefore lipomas are most common in those areas where the least fat is secreted. It has been frequently suggested that there is a causal relationship between a single trauma and the development of lipomas. This relationship has not been demonstrated. It cannot be denied, however, that long-continued irritation, as, for example, when the part of the body is exposed to frequent pressure, may be an etiological factor.

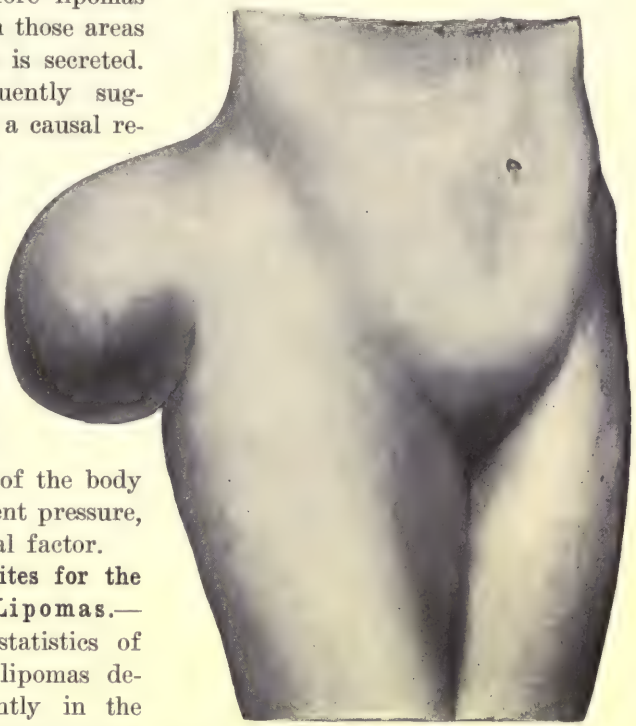


FIG. 303.—SUBCUTANEOUS LIPOMA IN THE REGION OF THE HIP.

Most Common Sites for the Development of Lipomas.—According to the statistics of Grosch and Stoll, lipomas develop most frequently in the subcutaneous connective tissues of the shoulder and back. They are less common upon the front and back of the neck, in the breast, and in the gluteal region, and are but rarely encountered upon the face, scalp, scrotum, and labia.

Subcutaneous Lipoma.—Subcutaneous lipomas sometimes appear as small, flat, at other times as nodular, lobulated growths, the size of a fist or a man's head. Sometimes a lipoma becomes so heavy during its later growth that its broad base is drawn out, forming a relatively narrow pedicle (lipoma pendulum). An œdema then often develops as the result of stasis, and when the tumor is incised fluid may be pressed out as from a wet sponge.

The skin covering a lipoma is of normal appearance, movable, and may be raised in folds. It may feel thick or thin, depending upon the amount of subcutaneous fat. If the skin is made tense, the shallow depressions between the different lobules of the tumor may be distinctly seen, especially if the tumor is immediately adjacent to the skin.

A lipoma may cause pain by pressure upon nerves. Multiple, symmetrical lipomas are frequently painful because of their relation to the connective tissues of the cutaneous nerves. Lipomas may be so situated or become so large that they cause considerable discomfort. Usually, however, they give rise to but few symptoms, and it is not at all uncommon to see a patient who has carried an enormous lipoma about for a number of years.

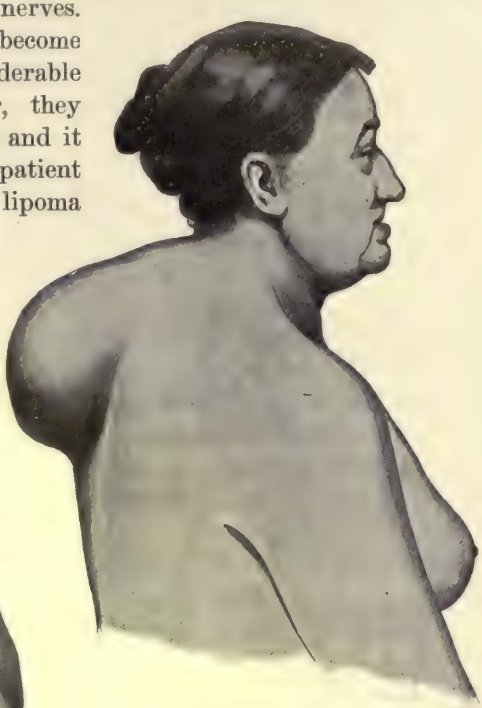


FIG. 304.—SUBCUTANEOUS AND PARTLY INTERMUSCULAR LIPOMA OF THE BACK.

FIG. 305.—SUBCUTANEOUS LIPOMA WHICH HAS BEEN GROWING GRADUALLY FOR FIFTEEN YEARS. The skin covering the tumor is very œdematous.

Fascial and Aponeurotic Lipomas. — As compared with subcutaneous lipomas, those developing within fasciæ and aponeuroses and intermuscular connective tissue are not common. These different forms are prone to develop in particular regions of the body, and a knowledge of the most common sites aids considerably in making a diagnosis.

The small, round, rarely lobulated lipomas occurring about the head are most common in

the frontal region, and are often referred to as *pericranial lipomas.* They lie beneath the aponeurosis or muscular fibers of the occipitofrontalis, and are quite firmly attached to the latter. They may produce a pressure atrophy of the periosteum and external plate of the subjacent bone. In this way a depression is made in the bone in which they lie, and they resemble closely in some cases dermoid cysts (von Bergmann).

Lipomas in the palm of the hand develop most frequently beneath the palmar fascia. They then extend between the metacarpal bones to reach the dorsal surface of the hand (Steinheil). Occasionally lipomas develop upon the fingers, especially upon the palmar surface, being either subcutaneous or attached to the bone.

Fig. 306.—Lipoma of the Forehead.

Subfascial lipomas also occur in the neck, back, and abdominal wall. They send off large processes between the muscles. Intermuscular lipomas may be encountered in the back, beneath the pectoralis major, upon the extremities, and in the abdominal wall. Some of the lipomas of the cheek, developing from the sucking pad, and of the tongue are intermuscular forms; others develop from the submucosa.

Lipomas Developing within the Abdominal Cavity.—The abdominal cavity is another but not frequent site for the development of lipomas. A portion of omentum which has been retained in a hernial sac for a number of years may proliferate to form a lipomalike mass (omental lipoma). The appendices epiploicæ at times become so large that they resemble a tumor, and they may become detached, forming free bodies in the peritoneal cavity. Small submucous lipomas occur in the stomach. The largest fatty tumors develop from the retroperitoneal tissues from which growths weighing from twenty to fifty pounds have been removed, sometimes successfully (statistics of Heinricius). Lipomas

of the mesentery are situated either at its root or along its intestinal attachment. Subserous lipomas, developing from the properitoneal fat and extending into the femoral and inguinal canals and through the foramina in the linea alba, dilate these openings and draw a funnel-shaped process of peritoneum after them. [Rose and Linhart have emphasized the relationship between hernia and subserous lipomas. Undoubtedly they are an etiological factor in a number of cases. They dilate the canal and draw the peritoneum, which forms the sac after them. Often in operating for a small hernia of the linea alba in the early stages a small subserous lipoma will be found, unassociated as yet with any definite sac.]

Lipomas of the Different Viscera.—Lipomas are rare in the different viscera, being most common in the kidney. The small tumors which develop in the latter never become larger than a walnut, and are usually situated in the cortex. They often contain smooth muscle fibers. These tumors are very rare in other organs, such as the lungs, liver, heart, uterus, and breast. Small lipomas developing from the pia mater are occasionally found at the base of the brain.

Lipoma Arborescens.—Lipoma arborescens is a particular form resulting from the proliferation of synovial villi and associated with the formation of clublike processes. This form of lipoma is associated with different chronic inflammatory processes in the joints, especially of the knee joint, where it was first observed by Joh. Müller. It is occasionally found in chronically inflamed tendon sheaths, especially in those of the hand (Stieda, Schmolk, Haeckel).

It should be mentioned that lipomas occasionally develop in the orbit, spermatic cord, tongue, and retromammary tissues.

Diagnosis.—The diagnosis of superficial lipomas is rarely difficult. It is based upon the position of the tumor, its slow growth and well-defined boundaries, mobility, lobulated structure, pseudo-fluctuation and consistency, which is sometimes soft, sometimes hard. Usually the painful lipomas are symmetrical, flat, and somewhat nodular, and may be easily differentiated from the round, spindle-shaped fibromas developing upon the nerves. If the surface of the tumor cannot be palpated and its boundaries cannot be definitely defined, the diagnosis of a benign tumor may be made, but frequently nothing definite can be said concerning the variety. In doubtful cases, cystic formations (dermoids, echinococcus cysts, hygromas) and tuberculous abscesses may be excluded by aspiration. A lipoma of the forehead differs from a periosteal gumma by being more mobile. Large lipomas of the peritoneal cavity may be regarded, because of their hardness, as fibromas or fibrosarcomas, the soft form as an ascites or an encapsulated tuberculous abscess. It is often difficult to differentiate between a retromammary

lipoma and a lipoma within the breast proper, between a lipoma beneath the parotid gland and one within it.

Technic of Removal.—Usually *the removal of a fatty tumor* is not difficult. [The tumor should be seized between the thumb and index

Fig. 307.—Diffuse Symmetrical Lipomas.

finger of the left hand and the skin made tense. An incision is then made down to the capsule and often the tumor fairly pops out. If any difficulty is encountered the tumor may be seized with sharp hooks or retractors and pulled from its bed, the dissection being completed with

the fingers, thick septa being divided with scissors.] In the excision of large tumors a sufficient portion of the thinned or œdematous skin covering the tumor should also be removed.

Diffuse Lipomas.—Clinically, a diffuse lipoma is differentiated from the circumscribed, encapsulated form. These diffuse lipomas may be congenital, being associated with partial giant growths of the extremities, or may occur as multiple, symmetrical growths in middle-aged people. They surround the neck as broad nodular growths (the so-called fatty neck of Madelung) and occur upon the trunk and extremities as transverse and oblique masses (Fig. 307).

These diffuse lipomas, described by Billroth as *lipomatosis regionaria,* are not tumors strictly speaking, but are merely localized accumulations of fat, secondary to an excessive development of fat in the individual. [They usually develop in people who drink large quantities of beer and eat to excess, and differ from true lipomas in that they decrease in size when beer is withdrawn and the patient is compelled to take exercise.] They bear the same relation to polysarcia and obesity that lobulated fibroma formation does to elephantiasis (Virchow).

The subcutaneous fat is chiefly involved by these growths, but they may extend deeper, passing in between the muscles. Sometimes it is necessary to remove these growths, especially when they occur in the neck, as they may press upon the trachea and interfere with breathing. A complete operation can never be performed, however, as the growths are not encapsulated. The tumors do not recur even after incomplete removal.

Repair of the sutured wound is often delayed by the discharge of a serous fluid, containing fat, which continues for some time. This fluid may even seep out through the stitchholes.

Literature.—*Blaschko.* Erbliche Lipombildung. Virchows Arch., Bd. 124, 1891, p. 175.—*Ehrmann.* Ueber multiple symmetrische Xanthelasmen und Lipome. Beitr. z. klin. Chir., Bd. 4, 1889, p. 341.—*Grosch.* Studien über das Lipom. Deutsche Zeitschr. f. Chir., Bd. 26, 1887, p. 397.—*Haeckel.* Lipoma arborescens der Sehnenscheiden. Zentralbl. f. Chir., 1888, p. 297.—*Heinricius.* Ueber retroperitoneale Lipome. Deutsche Zeitschr. f. Chir., Bd. 56, 1900, p. 579 and Arch f. klin. Chir., Bd. 72, 1904, p. 172.—*Köttnitz.* Ueber symmetrisches Auftreten von Lipomen. Deutsche Zeitschr. f. Chir., Bd. 38, 1894, p. 75.—*Langer.* Zur Kasuistik der multiplen symmetrischen Lipome. Arch. f. klin. Chir., Bd. 46, 1893, p. 899.—*Madelung.* Ueber denn Fetthals. Arch. f. klin. Chir., Bd. 37, 1888, p. 106;—Exstirpation eines vom Mesenterium ausgehenden Lipoma oedematosum myxomatodes mit partieller Resektion des Dünndarmes. Berl. klin. Wochenschrift, 1881, p. 75.—*Hellmut Müller.* Ueber die Lipome und lipomatösen Mischgeschwülste der Niere. Virchows Arch., Bd. 145, 1896, p. 339.—*Payr.* Beitr. z. Lehre von den multiplen und symmetrischen Lipomen. Wien. klin. Wochenschr., 1895, p. 733.—*Preyss.* Ueber die Operation der diffusen Lipome des Halses. Beitr. z. klin. Chir., Bd. 22, 1898, p. 469.—*Schmolk.* Zwei Fälle von Lipoma arborescens genu. Deutsche Zeitschr. f. Chir., Bd. 23, 1886, p. 273.—*Steinheil.* Ueber Lipome der Hand

und Finger. Beitr. z. klin. Chir., Bd. 7, 1891, p. 605.—*Stieda.* Lipoma arborescens. Beitr. z. klin. Chir., Bd. 16, 1896, p. 285.—*Stoll.* Beitr. z. Kasuistik der Lipome. Beitr. z. klin. Chir., Bd. 8, 1892, p. 597.

CHAPTER III

CHONDROMAS

Tumors which are composed of cartilage are called chondromas. Those chondromas occurring in parts which normally contain no cartilage were called enchondromas by Virchow to differentiate them from ecchondromas, which develop in parts normally containing cartilage. Enchondroma is therefore synonymous with heterologous chondroma. Hyperplastic cartilaginous growths are known as ecchondroses, but it is often impossible to make a sharp distinction between tumorlike and hyperplastic growths of cartilage.

Appearance and Histology.—Chondromas are nodular, soft, or hard tumors of opalescent appearance resembling normal cartilage. They are often multiple and may appear in great numbers. Usually they

Fig. 308.—Multiple Enchondromas of the Bones of the Hand.

Fig. 309.—Enchondroma of the Thumb.

develop slowly, but sometimes begin to grow rapidly and become quite large. These tumors are most common in young people.

Histologically they differ from normal cartilage, as the cells frequently do not possess a capsule, are less regular in shape, being oval,

round, fusiform, and stellate, and are not arranged according to any definite plan. The ground substance consists of hyaline, elastic, or fibrous cartilage. The different nodules composing the tumor are held together by vascular connective tissue, which may even penetrate into the cartilage. Fibrous, myxomatous, osteal, and angiomatous tissue may develop at the same time that the cartilage does, and mixed tumors, such as fibrochondromas, chondromyxomas, and osteochondromas are formed. As a result of the excessive proliferation of the cartilage cells the tumor may become transformed into a sarcoma (chondro-sarcoma, or if bone is also present into an osteochondro-sarcoma).

FIG. 310.—ROENTGEN-RAY PICTURE OF A CORTICAL ENCHONDROMA.

FIG. 311.—ENCHONDROMA OF THE SECOND METACARPAL BONE AS IT APPEARS EXTERNALLY AND UPON SECTION.

Method of Growth.—The growth of chondromas is sometimes expansive, sometimes infiltrating. Depending upon the character of their growth, these tumors are sometimes benign, sometimes malignant. If the tumor is surrounded by a layer of tissue resembling perichondrium, it merely displaces the neighboring structures as it grows. In the soft, cellular forms a capsule is frequently wanting, and then the cartilage cells grow into the spaces of the adjacent tissues, invade the veins and lymphatic vessels, and are carried to the lungs and lymphatic nodes. A continuous growth extending from a chondroma of the vertebra through the large veins to the heart has been observed (Ernst).

Changes Occurring in Chondromas.—Ossification (ossifying chondroma) is the most important of the changes which a chondroma may undergo. It is preceded by vascularization, as in normal bone forma-

tion, and finally the cartilaginous tumor is transformed into a bony tumor, the only indication of its cartilaginous origin being a thin covering of cartilage.

Regressive changes, such as calcification, myxomatous softening, and cyst formation, are common in chondromas. They are usually secondary to nutritional changes in the tumor tissue. If myxomatous softening occurs, a *chondroma myxomatodes* develops; if cyst formation a *chondroma cysticum*. Occasionally ulceration of the skin, the result of pressure, is observed. Putrefaction of the tumor may follow ulceration.

Most Common Sites of Chondromas.—Chondromas are most common in the bony system. Enchondromas developing in bone may be congenital; frequently they develop during the first two decades of life. They occur most frequently upon the phalanges, metacarpal and metatarsal bones as single or multiple tumors. A favorite locality for enchondromas is the fingers, where they form characteristic, shapeless, nodular masses. These tumors develop more frequently from the metaphysis than from the diaphysis (Nasse), from the interior than from the surface of bone. The bone surrounding one of these tumors undergoes pressure atrophy and a cortical enchondroma produces deep depressions in the surface of the bone involved, while a central enchondroma gradually destroys the bone as it grows, so that finally it is covered only by a thin shell of bone or periosteum. In the bone which is not destroyed small islands of cartilage may be found.

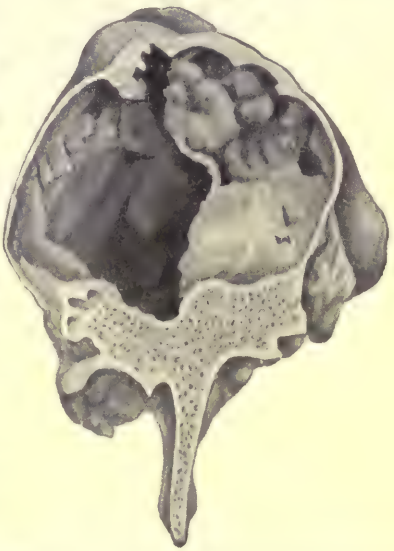


Fig. 312.—Cystic Enchondroma of the Scapula of an Adult.

In the long, hollow bones the metaphysis is the favorite site for the development of cortical and intraosteal chondromas. The phalanges when involved become considerably enlarged. These tumors may interfere with the growth of long bones, and when involved they become shortened and deformed, changes resembling closely those associated with rickets. In rare cases chondromas develop upon one side of the body only, and then the growth changes are unilateral (Ollier, A. Wettek). A central enchondroma may produce such an osteoporosis that a spontaneous fracture occurs. If the tumor has previously given rise to no symptoms and caused no enlargement of bone, it is often difficult to deter-

mine whether the fracture was secondary to the tumor or whether the tumor developed in the callus. Cystic softening of the central tumors, which are usually fibrochondromas, occasionally follows an injury and leads to the formation of large multilocular or coalescent cysts, the contents of which consist of a brownish fluid containing disintegrated blood and cholesterin. Often some of the original tumor tissue is found in these cysts, and the diagnosis as to their nature may be based upon this finding (Virchow, Schlange, Fritz König, and others). If no tumor tissue is present, islands of hyaline cartilage at some distance from the epiphyseal cartilage, and cartilaginous exostoses in the walls of the cysts indicate their origin and enable one to distinguish between them and the cysts (p. 749) occurring in osteitis deformans (Lexer).

FIG. 313.—CYSTIC ENCHONDROMA OF THE UPPER METAPHYSIS OF THE HUMERUS OF A CHILD NINE YEARS OF AGE, HEALED BY CURETTING OUT THE TUMOR TISSUE. Bony trabeculæ are still present between the nodules and cysts of the tumor.

Of the bones of the trunk those of the pelvis and the scapula are most often involved, being frequently the site for the development of very large tumors. Chondromas develop but rarely upon the ribs and skull bones, only occasionally upon the vertebræ, clavicle, sternum, and hyoid bone.

In many cases multiple cartilaginous exostoses are associated with enchondromas (Virchow, von Recklinghausen, Nasse, Läwen (Figs. 310 and 316).

Enchondromas are the result of some interference with normal bone formation, consisting either of a defect in the skeletal anlage, or of pathological changes occurring in bone during intra- or extrauterine life. The cause of the changes in normal bone formation is unknown. It is to be regarded as certain, however, that chondromas develop from germinal cartilaginous tissue which has been displaced from the epiphyseal zone (Virchow) into the bone marrow of the diaphysis. It is prob-

able that a number of these tumors originate from islands of cartilage which have been displaced from their usual position during an attack of rickets. Von Recklinghausen believes that the disturbance in bone formation is due to imperfect development of the blood vessels. The not infrequent association of multiple hæmangiomas with tumors of this character lends some support to the theory. The islands of cartilage cells displaced into the diaphysis may remain dormant or give rise to tumor formation later. If the tumor develops after a trauma, the latter is to be regarded as the exciting cause which stimulated the dormant island of cartilage cells to growth.

Chondromas are also encountered in organs and tissues which normally contain no cartilage, developing from cartilaginous rests which

FIG. 314.—BONE CYSTS IN THE HUMERUS OF A BOY FOURTEEN YEARS OF AGE. In spite of the fact that there was no tumor tissue, a diagnosis of a previous chondroma could be made because hyaline cartilage was found in the walls of the cyst and some medullary tissue was present. (Lexer.) Healing followed resection and transplantation of bone.

have been displaced during development. Cartilaginous tumors of the diaphragm develop from displaced portions of the skeletal anlage, of the ovary from portions of the primitive vertebræ, of the thyroid and

salivary glands and neck from portions of the cartilages of the branchial arches. Tumors about the ear, trachea, and bronchi develop from portions of the aural anlage, from tracheal and bronchial cartilages respectively. Pieces of cartilage which have not proliferated are sometimes found in the skin of the cheek (auricular appendages) being derivatives of the aural anlage. Remains of the branchial cartilages are also found in the tonsils, parotid glands, and the lateral regions of the neck. Reichel observed a chondroma in the capsular ligament of the knee joint which had developed from a synovial villus containing cartilage. Riedel has encountered chondromas in the wrist joint, Langemak in tarsal joints, Paulet and Honsell in different muscles (masseter and deltoid).

FIG. 315.—ENCHONDROMAS OF THE UPPER METAPHYSES OF THE BONES OF BOTH THIGHS ASSOCIATED WITH EXOSTOSES OF THE LOWER ENDS OF THE BONES (ELEVEN-YEAR OLD BOY).

Some of the cartilaginous growths occurring in the salivary and mammary glands, ovary, kidney, and uterus are of a complicated structure and are not to be classified with simple chondromas. The rare, multiple new growths occurring as small, hard nodules upon the inner side of the larynx and trachea; as larger growths, often becoming the size of an apple, upon the costal cartilages; as small flat projections upon the posterior surface of the symphysis and upon the intervertebral disks—are known as *ecchondromas.*

Diagnosis.—The diagnosis of the common chondromas is not difficult. The appearance of these growths, occurring as nodular and usually hard, painless, circumscribed tumors firmly attached to the bone and covered by normal or thinned skin which is not adherent, is quite characteristic. Mistakes in diagnosis should not be made when a number of these tumors are situated upon the bones of the hands and feet, and when there are a number of cartilaginous exostoses upon the metaphyses of the long bones. Small, isolated cortical chondromas resemble exostoses, but X-ray pictures enable one to differentiate between them (Fig. 310).

Central chondromas of the metaphyses of long bones may be mistaken for chronic inflammatory foci or myeloid sarcomas. Examination with the X-rays makes a differential diagnosis possible. The bone surrounding an inflammatory focus becomes thickened and sclerotic, casting a heavy shadow, while bone covering a central tumor becomes expanded and thin, casting a faint shadow. Distinct, bony trabeculæ may be seen between the different nodules or cysts of a central chondroma, while a myeloid sarcoma casts a shadow of the same density throughout (Fig. 313).

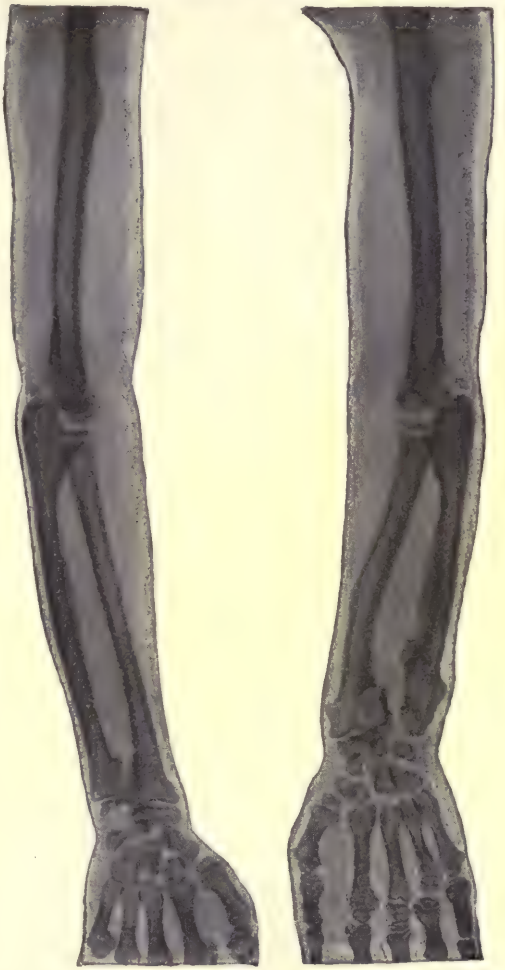


Fig. 316.—Enchondromas and Exostoses of the Lower Ends of the Bones of the Forearms. The bones upon the right side are shortened and deformed. Exostoses may also be seen upon the diaphysis of each humerus.

Chondromas of the soft tissues are to be recognized by their nodular form, slow growth, distinct, sharp boundaries and position. Sometimes

it is impossible to differentiate between chondromas occurring in the soft tissues and mixed tumors.

Indications for Treatment—Technic.—Because of their doubtful character, chondromas should be removed when so situated that the operation will not be dangerous or mutilating. Single tumors especially should be removed; if multiple, only those which grow rapidly or because of size or position give rise to symptoms.

If the bone involved has not already been destroyed, an attempt should be made to enucleate the tumor, preserving the continuity of the bone. Parts of the tumor left behind after enucleation should be removed with a curette. The deep cavities remaining in the bone after removal of the tumor may be filled with Mosetig-Moorhoff's bone plug (p. 254).

If the tumor rapidly recurs from pieces left at the previous operation or from previously latent germinal tissue, resection of the diseased bone may be indicated. Resection is also indicated whenever the destruction of the bone has been extensive. Amputation of the fingers or toes, hand or foot, is often necessary, because of the extensive destruction of the bones caused by these tumors. Enchondromas of the pelvic bones may become so large that they are inoperable.

CHAPTER IV

OSTEOMAS

Osteomas are tumors composed of bone. They are benign tumors which in their development may repeat periosteal as well as endochondral bone formation, and consist either of compact (osteoma durum or eburneum) or spongy bone (osteoma spongiosum), or contain large spaces filled with bone marrow (osteoma medullosum). Depending upon their origin, the surface of these tumors is covered with periosteum or cartilage (periosteal or fibrous, and chondral or cartilaginous osteomas).

Relation Between Osteomas and Hyperplastic Growths.—In no other class of tumors is it so difficult to make a sharp distinction between true tumors and hyperplastic growths. There is but little uniformity of opinion among authorities as to the basis upon which it should be made. This is so for the following reasons: (1) Bone is peculiar in that it reacts rapidly to inflammations and traumas of all kinds, proliferating to form tumorlike growths upon the surface of the part involved (inflammatory and traumatic exostoses); (2) there are a number of diseases of bone, such as osteitis deformans of Paget, osteitis fibrosa of

Recklinghausen, leontiasis ossea of Virchow, the cause and nature of which are unknown that are associated with the formation of tumorlike growths in bone. Bony growths, very closely resembling tumors, also develop in muscles. (3) A number of hyperplastic growths which genetically have no relation whatever to osteomas undergo secondary ossification, or the degenerated areas in them become calcified, so that it is often impossible to separate them from true tumors. A genetic relationship is found only in the ossifying fibromas and chondromas which develop from osteal tissues (fibroma and chondroma ossificans).

Most Common Site for Development.—Naturally osteomas are most common in bone, developing mostly as exostoses from the surface of bone; in rare cases as enostoses from their interior.

Exostoses.—Cartilaginous exostoses are more frequent than fibrous ones. Composed of spongy or compact bone, they occur as single or multiple tumors, often at a number of different points. They may be congenital or acquired, or may develop in early childhood. They grow slowly to attain the size of a fist. In rare cases they become larger, forming hard nodular tumors with a broad base or thin pedicle. They give rise to no symptoms unless they press upon adjacent nerves or become so large that they interfere with motion. Some of these tumors cease growing when the individual attains full growth, others continue to grow slowly but steadily even after this time.

Fig. 317.—Cartilaginous Exostosis of the Femur.

Most Common Sites for Development.—They are most common in the long, hollow bones developing near the articular ends and metaphyses. An exostosis which developed in early life is gradually displaced into the diaphysis as the bone increases in length. Frequently the bones become shortened and deformed; congenital defects may also occur. Exostoses are also found on the ribs, the clavicle, pelvic bones, and scapula; rarely on the bones of the fingers and toes, or those of the

skull and face. In other words, they may be found anywhere in the bony system where cartilage was originally present, and especially in the vicinity of the epiphyseal cartilages. In the pelvis they are most frequently found about the epiphyseal cartilages and along the crest of the ilium; on the scapula they occur upon the anterior surface and along the axillary border, often interfering with motion.

Multiple cartilaginous exostoses may give rise to a peculiar clinical picture. They may be associated with multiple chondromas, especially of the fingers, and with different anomalies of growth. Only one case (Chiari) which has been complicated by the development of a sarcoma has been reported.

Origin of Exostoses.—Cartilaginous exostoses develop from islands of cartilage which have been displaced as a result of defects in the skeletal anlage or of pathological processes occurring during the later development of the bone. The frequent multiple occurrence of these tumors, hereditary transmission, simultaneous anomalies in growth supposedly due to the same cause, their frequent association with chondromas which genetically are the same and are frequently found in one member of the family, while exostoses are found in the other members, and finally the demonstration of small displaced islands of cartilage near by the exostoses (von Recklinghausen, Chiari) all point to this origin.

Fig. 318.—Cartilaginous Exostosis of the Proximal Phalanx of the Third Finger.

Exostosis Bursata.—The exostosis bursata (von Volkmann) is a special form which is situated most commonly upon the lower epiphysis of the femur and is covered by a bursa similar to other bursæ occurring about the knee joint. The bursa is firmly attached to the edges of the cartilaginous covering of the nodular growth and contains a synovialike fluid, and in rare cases numerous free, cartilaginous joint bodies. Many regard the bursa as a secondary formation, the result of long-continued pressure or irritation (von Volkmann, von Recklinghausen); others believe that during the development of the exostosis

a part of the synovial membrane of the joint was evaginated and later became constricted to form a distinct bursa (Rindfleisch); while still others believe that the bursa develops from a portion of the joint anlage which became displaced during fœtal life (Fehleisen).

Diagnosis.—The diagnosis of cartilaginous exostoses is based upon their position and relation to the bone, their slow growth, hardness, and well-defined borders. The clinical picture of multiple cartilaginous exostoses is characteristic, and the diagnosis, as a rule, should be made without difficulties. Single tumors may be confused with periosteal fibromas and chondromas unless one makes use of X-ray pictures.

Fibrous Exostoses.—Frequently fibrous exostoses cannot be differentiated from circumscribed growths of bone not at all related to true tumors etiologically.

Relation to Inflammation and Trauma. — Inflammation and traumatism often stimulate the periosteum to the formation of large, rapidly growing, bony growths (Honsell). Sometimes tumorlike processes develop from the callus following a fracture and extend between the neighboring muscles, developing apparently from separated and displaced fragments of periosteum. Other exostoses develop where circumscribed areas of periosteum are subjected to constant pressure (subungual exostoses of the great toe, exostoses upon inner side of great toe in hallux valgus), and still others develop where tendons or muscles are inserted. They appear as roughened areas, bony projections and crests.

Fig. 319.—Cartilaginous Exostosis on the Medial Side of the Upper Metaphysis of the Tibia in Genu Valgum Rhachiticum.

Of the bones of the skull, the frontal and parietal are most frequently involved, the exostoses occurring as single or multiple, nipple or buttonlike, spinous and pedunculated tumors covered by a thin layer of periosteum. They may be situated upon the

internal or external plate of these bones or upon both plates, opposite each other. The exostoses rarely become larger than a walnut. One of the larger forms is represented in Fig. 322.

Only a part of the exostoses of the orbit and the different cavities of the face develop from periosteum, as the osteomas of the frontal and sphenoidal sinus develop from fœtal rests displaced from the cartilaginous anlage of the ethmoid (Arnold). If these tumors fill the cavity

Fig. 320.—Multiple Cartilaginous Exostoses of the Metaphysis of the Femur and Tibia with a Chondroma of the Upper Metaphysis of the Fibula on the Right Side.

in which they lie, they are called "*encapsulated bony bodies*" (Cruveilhier). If, as a result of suppuration and necrosis, their pedicle is destroyed and the connection with the wall of the cavity is lost, they are called "dead osteomas" (Tillmanns). These exostoses grow slowly, and gradually produce a pressure atrophy of the walls of the cavity in which they lie, extending to neighboring cavities or the surface; for

example, from the frontal sinus to the forehead or into the orbit, from the sphenoidal sinus into the cranial cavity.

Osteomas of the Jaws.—In the jaws, not including the encapsulated osteomas of the antrum of Highmore, there are found periosteal nodular exostoses which often attain considerable size and central tumors surrounding tooth buds. Only a part of the latter are pure osteomas; the remainder are odontomas, usually composed of dentine and developing from normal or displaced teeth.

Fig. 321.—Subungual Exostosis.

Symptoms.—The symptoms depend upon the position of the tumor and the direction in which it extends. Growing from the under surface of the skull or from the vertebræ, they may give rise to serious symptoms from irritation or compression of the brain and spinal cord. A tumor developing in the frontal sinus, by occluding the communication between the accessory sinus and the nose, may lead to sinus inflamma-

Fig. 322.—Enormous Exostosis of the Temporal Bone, Part of which Projects into the Skull Cavity. Sclerosis of the bones of the skull. (von Volkmann.)

tion. Tumors may also be so situated as to press upon important nerves (optic and trigeminal) or to cause ugly deformities of the face.

Diagnosis.—The diagnosis, because of the slow, painless growth and the circumscribed form of the tumor, or of the symmetrical expansion of the bone in osteomas of the accessory sinuses, is usually not difficult.

In the beginning they may resemble clinically central sarcomas. When an empyema of the frontal or maxillary sinuses develops secondary to the tumor, the inflammatory symptoms may be most prominent. The deep shadow which an osteoma casts in X-ray pictures aids, in doubtful cases, in making the diagnosis. The bony growths occurring in leontiasis ossea do not have sharp, distinct outlines.

Treatment.—The treatment consists of complete removal of the osteoma when possible. Recurrences may develop from pieces of the tumor which are left behind.

Myelogenous Enostoses.—True (myelogenous) enostoses of the long, hollow bones are exceedingly uncommon (Virchow, Bennecke). Those developing in the skull bones from the diploë and extending outward and inward cannot be differentiated from exostoses which develop on the surface and later perforate the bones (von Bergmann).

Bony Growths in Soft Tissues.—Bony growths also occur in the soft tissues. Even in these cases it is often difficult to distinguish between osteomas proper and inflammatory hyperplastic growths of connective tissue which have secondarily undergone calcification and ossification. Part of the small nodular osteomas of the brain, of the flat growths in the dura mater (falx cerebri), of the circumscribed foci occurring in the lungs, of the multiple small nodules and cords upon the inner surface of the trachea, and of the bony deposits in the cavernous tissue of the penis (penis bones) develop from displaced cartilaginous rests. This is especially true of the osteomas of the lung and trachea which develop from the cartilaginous anlage of the respiratory passages.

Myositis Ossificans.—The ossification of muscle gives rise to an important and peculiar clinical picture. Although the disease may be conveniently discussed in this chapter, the pathological changes are not exactly similar to those resulting in the formation of osteomas in soft tissues.

This disease may be progressive, affecting in succession a number of different muscles (*myositis ossificans progressiva*), or limited to one muscle (*myositis ossificans cimcumscripta*), the changes following a trauma.

In myositis ossificans progressiva a number of different muscles gradually change into bone. In some of the cases the changes begin in the periosteum (Virchow) leading to the formation of exostoses at the point of attachment of the muscle, and then extend to the connective

tissue of the muscles and to the fascia. In other cases the disease begins in the bellies of the muscles (Lexer, von Zoege-Manteuffel, Stempel) and extends in both directions to their points of attachment. The new bone which is formed may become attached to the bones at the point of attachment of the muscles or may remain free.

Histological Changes in Myositis Ossificans.—Histological examinations made in the early stages of the disease (Lexer, Stempel) have revealed germinal tissue rich in cells which infiltrates the intermuscular connective tissues (perimysium externum and internum), causing a pressure atrophy of the muscle fibers and bundles which are replaced by a firm connective tissue or bone, repeating in its development either the periosteal or endochondral type of bone formation. In the beginning of the cellular growths, round-cell infiltration, a change indicative of inflammation, may be observed.

These changes have some relation to the clinical picture, but cannot be regarded as either the cause or the result of the process. The proliferation extends from the perimysium to the tissues surrounding the tendons and to the fasciæ, and both become ossified.

Up to the present time about fifty cases of this disease have been observed. It begins in early life, rarely after the twentieth year, and is about three times more frequent in the male than in the female. There are no data concerning heredity.

Fig. 323.—Progressive Ossifying Myositis from a Photograph Owned by Professor Helferich.

Muscles Most Frequently Involved and Clinical Course.—The disease usually begins in the muscles of the neck and back. Suddenly the muscles or groups of muscles involved swell and become painful. The swelling is sometimes accompanied by fever and a slight reddish or bluish discoloration of the skin covering the muscles. The pain following motion and pressure gradually subsides, and the swollen muscles, of a doughy consistency at first, become smaller and indurated. This stage has been called by Münchmeyer the stage of connective-tissue indura-

tion. In some cases the pathological changes do not progress farther. In the majority of cases, however, the changes progress, and corallike, scalloped cords and plates of bone develop throughout the muscle primarily involved and extend to adjacent muscles. These not only destroy the function of the muscles involved, but fix the extremities in uncomfortable and useless positions, as the newly formed bone bridges over the joints, uniting bone with bone.

A number of years may intervene between attacks, in each of which new muscles are involved. Finally the entire musculature of the trunk, different muscles of the extremity, and the muscles of mastication may become ossified. The patient then becomes transformed into a motionless mass (ossified man). Deglutition and respiration gradually become more and more difficult, and finally after a number of years the patient dies of exhaustion or of aspiration pneumonia.

Cause of the Disease.—There has been a great deal of discussion as to the cause of the disease. The name myositis ossificans has been given it because of the inflammatory symptoms in the muscles at the beginning of the disease. It is well established that a very cellular germinal tissue develops in the intermuscular spaces which becomes transformed into dense connective tissue and bone. It has been suggested that the disease is the result of a congenital anomaly of development in the skeletal system, or, as Stempel thinks, is due to the imperfect differentiation of the mesenchyme, as a result of which the muscles acquire the property of forming bone. In some cases malformations of the fingers and toes (microdactylism, anchylosis of the phalanges, absence of the terminal phalanges and muscles) have been present at the same time (Virchow, Helferich), supporting the view that the disease is a congenital anomaly of development.

The *diagnosis* of the disease may be difficult at the beginning.

Treatment.—Treatment has no influence whatever upon the course of the disease. The patient should be made as comfortable as possible, and pieces of bone which interfere with motion or render the patient uncomfortable (especially bone about the jaw interfering with mastication and about the joints) should be removed.

Myositis Ossificans Circumscripta.—Myositis ossificans circumscripta is not a progressive lesion. It follows repeated traumatisms or a single severe injury, and remains limited to the muscle primarily involved, in which histological changes (similar to those described above in myositis ossificans progressiva) occur. The bony plates and cords which develop lie firmly imbedded in the connective tissues of the muscle or become fused with the bone at its points of attachment.

The so-called rider's bone develops in the adductor muscles, more rarely in the pectineus and gracilis, after injuries received during

horseback riding; the so-called exercise bone develops in the deltoid muscle after injuries caused by the rifle coming forcibly in contact with the muscle during drill. The turner's bone, which develops in the brachialis anticus, is a rarity. The development of bone in these cases is probably due to some congenital anomaly in the connective tissues of the muscle, as the result of which they acquire the property of forming bone when injured or irritated.

The ossification of muscle following a single trauma (*myositis ossificans traumatica*) gradually develops in the course of months. The brachialis anticus and quadriceps extensor muscles are most frequently involved. Lymph cysts may also develop in the connective tissues surrounding the piece of bone (Wolter). Myositis ossificans traumatica follows severe contusions, dislocations, and fractures, and cannot be separated from those bony growths which extend from an exuberant callus into the soft tissues. The bone probably develops in these cases from separated and displaced fragments of periosteum.

The position of the bone in a muscle which is exposed to repeated traumas or has been severely contused suggests at once the diagnosis. The findings may be readily verified by an X-ray picture. [Gouty deposits in muscles and about joints are sometimes confusing.]

Although cases have been observed in which the bone disappeared spontaneously and in which massage has been helpful, extirpation with subsequent suture of the wound in the muscle is to be recommended if there are symptoms.

LITERATURE.—*v. Bergmann.* Geschwülste der Schädelknocken. Handb. d. prakt. Chir., 2. Aufl., Bd. 1, p. 123.—*Bennecke.* Exostose der Tibia. Zentralbl. f. Chir., 1904, p. 500.—*Bornhaupt.* Ein Fall von linksseitigem Stirnhöhlenosteom. Arch, f. klin. Chir., Bd. 26, 1881, p. 589.—*Busse* and *Blecher.* Ueber Myositis ossificans. Deutsche Zeitschr. f. Chir., Bd. 73, 1904, p. 388.—*Chiari.* Zur Lehre von den multiplen Exostosen (mehr als 1,000 Exostosen und ein Spindelzellensarkom am Humerus). Prager med. Wochenschr., 1892, No. 35.—*Eckert.* Zur Kenntnis der Osteome des Unterkiefers. Beitr. z. klin. Chir., Bd. 23, 1899, p. 674.—*Fehleisen.* Zur Kasuistik der Exostosis bursata. Arch. f. klin. Chir., Bd. 33, 1886, p. 152.—*Honsell.* Ueber traumatische Exostosen. Beitr. z. klin. Chir., Bd. 22, 1898, p. 277.—*Lexer.* Das Stadium der bindegewebigen Induration bei Myositis ossificans progressiva. Arch. f. klin. Chir., Bd. 50, 1895, p. 1.—*Nadler.* Myositis ossificans traum. mit spontanem Zurückgang der Muskelverknöcherung. Deutsche Zeitschr. f. Chir., Bd. 74, 1904, p. 427.—*Nasse.* Ueber multiple kartilaginäre Exostosen und multiple Enchondrome. v. Volkmanns Samml. klin. Vorträge, No. 124, 1895.—*Reich.* Ein Beitrag zur Lehre über die multiplen Exostosen. Deutsche Zeitschr. f. Chir., Bd. 43, 1896, p. 128.—*Riethus,* Exost. bursata mit freien Knorpelkörpern. Beitr. z. klin. Chir., Bd. 37, 1903, p. 639.—*Rothschild.* Ueber Myositis ossificans traumatica. Beitr. z. klin. Chir., Bd. 28, 1900, p. 1.—*Schuler.* Ueber traumatische Exostosen. Beitr. z. klin. Chir., Bd. 33, 1902, p. 556.—*Stark.* Ueber multiple kartilaginäre Exostosen und deren klinische Bedeutung. Beitr. z. klin. Chir., Bd. 34, 1902, p. 508.—*Stempel.* Die sogenannte Myositis ossificans progressiva. Mitteil. aus d. Grenzgeb., Bd. 3, 1898.—*Virchow.* Ueber Myositis ossi-

ficans progressiva. Verhandl. der Berl. med. Gesellsch., 1894, I, p. 172 and II, p. 142 and 1900, I, p. 151.—*Wolter.* Ueber Myositis ossificans traumatica mit Bildung von Lymphzys' en. Deutsche Zeitschr. f. Chir., Bd. 64, 1902, p. 351.—*v. Zoege-Manteuffel.* Demonstration eines Skelettes mit Myositis ossificans. Chir.-Kongr. Verhandl., 1896, I, p. 43.

CHAPTER V

ANGIOMAS

HÆMANGIOMAS

THE tumors composed of abnormally arranged, tortuous, and dilated vessels are classified as angiomas. The term angioma, however, should be applied to those tumors only in which there is an actual new formation of vessels or a proliferation of the vessel walls, aneurysms and varicose veins being thus excluded. Angiomas composed of blood vessels (hæmangiomas) are distinguished from those composed of lymphatic vessels (lymphangiomas).

Three forms of hæmangioma are distinguished: Hæmangioma simplex, cavernosum, and racemosum.

Hæmangioma Simplex.—The hæmangioma simplex is also known as a telangiectasis. This term, however, does not fully describe the nature of the tumor, as there is not only "a dilatation of the vessels," but an actual new formation of vessels as well. During the removal of such a growth small, dark-red, spurting lobules may be seen at the edge of the tumor. These are held together by connective tissue, and may extend close to the epidermis or be covered by a thin layer of the cutis and reach into the surrounding fatty tissues, muscle, and fasciæ. Not infrequently these growths are surrounded by a delicate connective-tissue capsule which is united with the neighboring structures only at the points where the vessels enter and leave the growth.

The tumor is composed of dilated, interlacing capillaries and small vessels, the walls of which contain flat or cubical endothelium and circularly arranged connective-tissue bundles. If both the endothelium and connective tissue have proliferated (angioma simplex hyperplasticum of Virchow) and the lumina of the vessels have become narrow as a result, it is often difficult to differentiate the vessels from sweat and sebaceous glands, both of which may be found in these tumors. The transformation of the vessels into solid cords consisting of proliferated endothelium forms a transitional stage to the hæmangio-endotheliomas.

Angiomas are not infrequently combined with other forms of tumors

of the connective-tissue group, forming angiolipomas, angiofibromas, angiosarcomas, etc.

Simple hæmangiomas enlarge in the following way: the processes developing from the vessels extend like buds into the surrounding tissues, and by a continuous new formation and dilatation of the capillaries the adjacent tissues become completely infiltrated. This method of growth, which resembles the infiltrating growth of malignant tumors, leads to the destruction of the infiltrated tissue, even if it is bone. They never form metastases, however, and do not enlarge in this way if encapsulated. The growth, sometimes slow, at other times rapid, is frequently interrupted for long intervals or ceases permanently after a short time. Secondary changes following inflammation may cause complete involution.

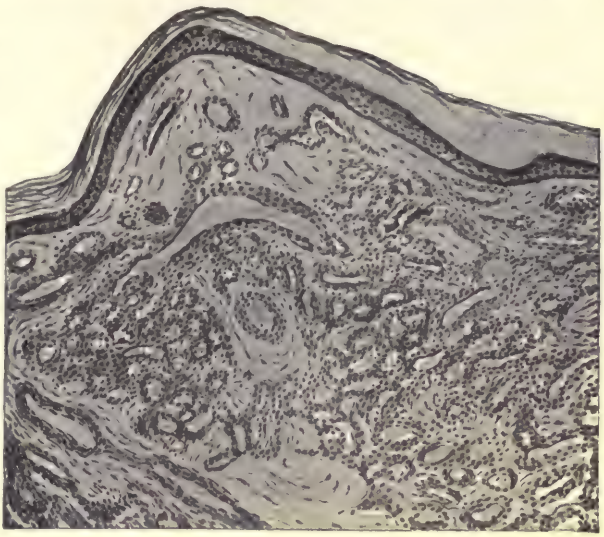


FIG. 324.—HÆMANGIOMA SIMPLEX CUTIS.

Simple hæmangiomas are the result of developmental anomalies. According to Ribbert, the area supplied by a small artery develops from the beginning without any connection with surrounding tissues, or loses its connection during subsequent growth and develops independently. The facts emphasized by Virchow that angiomas are commonly situated about the lips, cheeks, eyelids, and the root of the nose, the position of earlier fœtal clefts, and that they are frequently congenital and often multiple support this theory.

Simple hæmangiomas develop most commonly in the skin and subcutaneous tissues. Although any part of the body may be involved, two thirds of these tumors occur in the skin of the face.

Cutaneous Angiomas.—The cutaneous angiomas may be present at birth, appearing as light-red or dark-red, well-defined, round, or scalloped blotches or elevations, or they may develop during the first few weeks or months of life from small, scarcely noticeable points (resembling a flea bite) from which little delicate vessels radiate (nævus vasculosus, flammeus, port-wine mark). Some of these nævi grow very

rapidly, extending within half a year over the entire half of the face; others enlarge slowly, their growth corresponding to that of the body. Not infrequently the veins of the surrounding tissues become dilated.

If the tumor also extends into the deeper tissues, nodular, polypoid, and lobulated masses of dark-red color covered by a delicate skin develop which resemble the changes (*vide* p. 793) found in elephantiasis (elephantiasis hæmangiectatica).

Subcutaneous Angiomas.—The subcutaneous appear later than the cutaneous angiomas, remaining concealed beneath the skin until the latter becomes thin enough to allow the bluish shimmer to show through. A distinct swelling produced by filling of the vessels, when the patient cries, or the principal vessels are pressed upon and the circulation interfered with, and the extension of the growth to the epidermis leading to the formation of red blotches and elevations as in cutaneous angiomas indicate the presence of these tumors.

If both the skin and subcutaneous tissues are involved, pads and folds form upon the flat surface of the angioma from which, especially upon the lips, lids, and nose, large, purple, blackberrylike, lobulated, and pedunculated tumors may develop (Fig. 326). These tumors contain parts which are transitional to the cavernous angiomas.

Fig. 325.—Simple Cutaneous and Subcutaneous Hæmangioma.

A simple angioma occurring upon the lips may produce a macrocheilia similar to that caused by a lymphangioma. The upper lip, which is more frequently involved, then hangs down as an irregular bluish fold over the mouth, while the lower lip when involved projects outward like a snout. The tumor enlarges when the patient stoops, cries, or becomes excited. It has been said of Emperor Leopold, that when angry his lip, the seat of such a growth, hung down to his chin.

Angiomas of the eyelid, which may be primary in these structures or extend to them from the temporal and naso-frontal regions, should be mentioned, as they may invade the orbit and threaten the integrity of the eye.

Simple angiomas of the scalp, according to Heinecke, comprise thirty-three per cent of the angiomas of the head. They occur most frequently in the frontal region, developing about the glabella, at the inner extremity of the eyebrow, and over the fontanelles.

Changes Which May Occur in an Angioma.—The most important changes which may occur in a simple angioma are inflammation, hæmorrhages, and spontaneous involution. It has often been observed that not only the small, but also the larger flat angiomas may completely or partially disappear following the obliteration of the vessels composing them.

Fig. 326.—Simple Lobulated Hæmangioma.

Frequently inflammation, which develops after an injury of the thin, easily vulnerable skin, precedes and favors subsequent ulceration, cicatrization, and obliteration of the vessels. It has occasionally been attempted to cure angiomas in children by infecting them. Hæmorrhages are not frequent. When they occur they are apt to be profuse, but may easily be controlled by a bandage exerting mild pressure.

Simple angiomas are also found in rare cases in fat, especially in that of the orbit, in muscles, the breast, in bone, the brain, and spinal cord. The small, macular, and wartlike hæmangiomas, never becoming larger than a pea, which occur as multiple growths upon the surface of the body associated with soft and pigmented warts, form a special group. They develop usually in old age.

Hæmangioma Cavernosum.—The cavernous angioma (hæmangioma cavernosum) resembles in structure the corpus cavernosum, being composed of retiform blood spaces. Frequently transitional stages to the cavernous hæmangioma are found in the simple hæmangioma, from which they may develop. The irregular cavities, filled with blood and communicating with each other, are surrounded by a network of fibrous tissue containing elastic fibers. The thickness of the network varies in different parts of the tumor. These tumors are nourished by a single artery and discharge their blood into dilated veins. Thrombosis of the

blood spaces leads to localized connective-tissue changes. If the thrombi become calcified, phleboliths are formed. Single spaces may become closed and transformed into blood cysts in this way. These tumors may be well encapsulated or the capsule may be entirely or partly absent, and then the tumor extends without any sharp line of demarcation into the surrounding tissues. The growth, as in simple angiomas, may be partly expansive, partly infiltrating, slow but continuous, or rapid after remaining stationary for some time. An encapsulated tumor frequently ceases to grow. Involution may be complete after thrombosis and cicatricial contraction of parts of the tumor.

The fact that these tumors are frequently congenital and multiple indicates that they, like simple angiomas, are the result of some developmental anomaly, the exact nature of which is unknown.

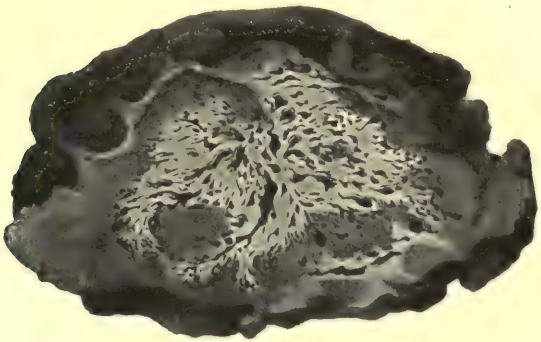


FIG. 327.—CAVERNOUS HÆMANGIOMA OF THE SUBCUTANEOUS FAT (AFTER EXTIRPATION).

FIG. 328.—CAVERNOUS HÆMANGIOMA OF THE EAR.

Most Common Sites for Development.—These tumors develop most frequently in the skin and subcutaneous tissues, being most common in the cheeks, eyelids, lips, and scalp. Other parts of the body are more rarely involved and with about the same frequency. These tumors, which may be present at birth, or develop shortly after, more rarely in later life, assume a number of different forms. Cavernous angiomas of the skin are characterized by the formation of dark-blue, bluish-black, nipplelike, and nodular growths, or of large, blackberrylike, lobulated masses covered by a delicate epidermis; cavernous angiomas of the subcutaneous tissues by the formation of a flat swelling, covered by slightly bluish, discolored, otherwise normal skin, or by skin the seat of a simple hæmangioma or nipplelike growths, such as are associated with cavernous hæmangiomas of the skin. If both occur together, folds in skin, such as are found in elephantiasis (elephantiasis cavernosa), may develop. Cavernous hæmangiomas of the cheeks, eyelids, and

lips soon extend to the mucous membranes, forming bluish nodules and lobulated growths.

Cavernous angiomas of the face frequently assume rapid growth, extending from the cheek to the mucous membranes of the mouth cavity and lips, finally involving the entire half of the face and head. If they occur in the scalp, the spongy tissue composing the tumor may grow through the bones and become connected by means of dilated emissary and larger veins with the venous sinuses. Excruciating pain may be produced if the tumor presses upon nerves, especially upon the branches of the trigeminal nerve. A cavernous angioma of the orbit may displace the bulb of the eye and threaten its integrity.

It should be mentioned that these tumors occur in the tongue and occasionally in the different muscles (muscles of the calf, rectus abdominis, sterno-cleido-mastoid, and masseter).

Of the abdominal viscera, the liver is most frequently the seat of cavernous tumors, occurring as small, multiple growths which are accidentally found during post-mortem examinations. Sometimes they appear as large, occasionally pedunculated, tumors, especially in children. Cavernous tumors may also occur in the spleen, bone marrow, uterus, and intestines, but are very rare.

Hæmorrhage, following rupture of nodules covered by thin skin, may be quite profuse, but is easily controlled by a bandage exerting mild pressure and by cauterization.

Diagnosis.—The diagnosis of hæmangiomas, the simple as well as the cavernous, is based upon their peculiar color and form and upon the fact that they can be emptied by pressure, but refill when the pressure is removed. The soft tumor masses, nodular if thrombi or phleboliths are present, become smaller and less discolored when pressure is exerted, but rapidly fill with blood, assuming their original form and color as soon as the pressure is released. In tumors of the dependent portions of the body the increase in size is very noticeable when pressure is exerted upon the veins passing from the tumor, or when the patient stands up. After compression of a subcutaneous angioma of the scalp, the pits and fissures in the bone through which the dilated, anastomosing veins pass to the interior of the skull may be easily felt. Only the deep-lying, calcified, or mixed tumors (lipoangioma) do not decrease in size upon pressure. Cavernous hæmangiomas may pulsate if nourished by large arteries. The clinical picture of angiomas is characteristic, and the diagnosis usually not difficult. Sometimes it is difficult to differentiate the isolated, subcutaneous forms from other tumors, especially when there is no involvement of the skin. It is not difficult to exclude plexiform angiomas, aneurysms, and varicose veins if the symptoms are pronounced.

Treatment.—There are a number of methods which may be employed in the treatment of simple and cavernous angiomas.

Superficial and deep angiomas, when encapsulated, should be excised. If the tumor is situated upon the extremities, hæmorrhage should be controlled during the operation by elastic constriction; if situated upon other parts of the body, by digital compression or special instruments. If the tumor is not encapsulated and extends into the deeper tissues, the greater part of it should be rapidly removed with a sharp spoon (von Bergmann), for after the large blood spaces are destroyed there is but little hæmorrhage from the small arteries entering the tumor tissue. Large defects should be skin-grafted or closed by plastic operations.

Deep angiomas of the face extending through the cheek to the mucous membrane, and angiomas which perforate the bones of the skull should not be excised. In treating angiomas in young children, the bloodless methods are always to be preferred when they can be employed. In the treatment of superficial birthmarks, a single application of fuming nitric acid is a simple and effective method. The eschar produced by this acid extends deeply enough, and when it is cast off a delicate scar remains. The skin surrounding the angioma should be protected from the acid by adhesive plaster or some other device.

Puncturing with the actual cautery (ignipuncture) is often successfully employed in the treatment of elevated nodular nævi, which should never be excised when occurring in weak children. It may be necessary to repeat ignipuncture a number of times, as the tumor cannot be destroyed by one application without leaving unsightly deformities, and it recurs from the tissue which is not destroyed unless the treatment is repeated after an interval. The scars resulting from this treatment are large and unsightly, and therefore this method cannot be employed for angiomas of the face. Aseptic dressings should be applied after separation of the eschar to prevent secondary infection, for infections developing in hæmangiomas of the cheek or scalp may easily extend to the meninges. Payr, after making small incisions in the skin, has inserted small pieces of magnesium into the tumor in all directions. As the metal is being absorbed the blood coagulates, the large dilated vessels become occluded, and even deep extensive tumors sometimes disappear. This method is especially suited for the treatment of inoperable hæmangiomas of the skull and face.

The injection of chemicals sometimes cause a gradual obliteration of the vessels and reduction in the size of the tumor. Alcohol has been recommended by Schwalbe. From fifteen to sixty drops of seventy or eighty per cent alcohol are injected at first into the margins, later directly into the tumor, some days intervening between each injection.

The injections should not be made where the skin is thin, as it may become necrotic, favoring hæmorrhages and infection. [Repeated injections of small quantities (1 to 2 c.c.) of boiling water with a hypodermic needle and syringe directly into the angioma offers one of the best methods of treatment.]

Some of the flat and superficial hæmangiomas have been successfully treated by the X-rays (*vide* p. 774).

The Cirsoid Aneurysm.—The cirsoid angioma (cirsoid aneurysm, angioma arteriale racemosum of Virchow) consists of thickened, dilated, tortuous pulsating vessels, the arteries supplying the tumor emptying directly into large blood spaces without the intervention of capillaries. The dilatation of the vessels may be fusiform or saccular. Frequently the afferent artery is thickened for a considerable extent as the result of proliferation of its walls, while the efferent veins, communicating as they do directly with the artery, are transformed into large, pulsating cords. For this reason this form of angioma is frequently referred to as a phlebarteriectasia.

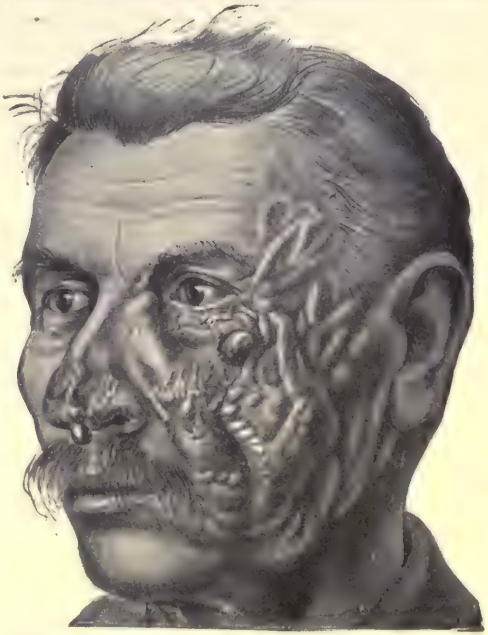


Fig. 329.—Cirsoid Aneurysm of the Face which was not Improved by Ligation of the External Carotid Artery and Other Large Branches Supplying it and by the Injection of Alcohol.

Cirsoid angiomas develop most frequently from simple, congenital hæmangiomas. They also develop after frequently repeated mechanical injuries (e. g., after pulling the ears, König) after injuries of the hand received while rowing, after a single trauma, and even without any apparent cause.

It is probable that this form of new growth is the result of some congenital defect in the arterial anlage, as a result of which the tissues composing the artery may be stimulated to proliferation by a number of different influences.

Most Common Sites.—Cirsoid angiomas occur most frequently in the scalp and face, more rarely in the extremities, the arm (hand and forearm) being next most frequently involved.

The superficial, tortuous, anglewormlike strands and masses are covered by a thin, cyanotic skin which is frequently adherent at a number of different points.

When they occur in the face, the skin covering the tumor is frequently the seat of a simple angioma, or is raised to form a flat swelling,

FIG. 330.—CIRSOID ANEURYSM OF THE HAND AND FOREARM (ENGLEBRECHT'S PREPARATION).

which gradually disappears into the surrounding tissues. The vessels composing the tumor pulsate, the pulsations being transmitted to the skin. Rhythmical impulses and thrills may be felt when the angioma is palpated; loud blowing and buzzing bruits, transmitted to communicating vessels, may be heard when the tumor is auscultated. In angiomas of the extremities these signs disappear when pressure is made upon the principal arteries. This does not always occur in the face, because the anastomosing arteries are more numerous; frequently, however, a slowing and strengthening of the pulse can be noticed, apparently the result of the diversion of the blood into the general circulation following the exclusion of so large a circulatory area.

FIG. 331.—RACEMOSE HÆMANGIOMA OF THE SCALP (VON LANGENBECK'S COLLECTION).

Clinical Course and Regressive Changes.—These tumors develop quite rapidly at first; later more slowly, growth often being interrupted by long intervals. When they occur upon the head the patient often complains of headache and dizziness; throbbing of the head and ringing in the ears, preventing sleep; excruciating pain

caused by pressure upon the nerves, and functional disturbances, the result of adhesions between the tumor and adjacent nerves and muscles.

Necrosis of the atrophic skin covering these growths is a serious matter, as chronic ulcers favoring hæmorrhage and infection then develop. Even the terminal phalanges of the fingers may become necrotic when the tumor involves the hand.

Diagnosis.—The diagnosis of cirsoid aneurysm is not difficult, as the appearance of the tumor composed of anglewormlike, tortuous, pulsating vessels is quite characteristic. It can scarcely be mistaken for any other lesion.

Treatment.—Treatment is very unsatisfactory. The ideal method, complete extirpation with ligation of the principal artery and innumerable smaller ones, is frequently impracticable, because of the size of the growth. Ligation of the principal artery alone has given no, or only transitory, results. In cirsoid aneurysm of the extremities this procedure is even dangerous, because of the possibility of gangrene.

In a male patient with an extensive cirsoid angioma of the left side of the face, whose picture is reproduced in Fig. 329, all the large, accessible arteries surrounding the tumor, such as the external carotid, the facial, the large branches of the temporal, and the angular at the inner canthus of the eye, were ligated without success.

Injection of chemicals with the object of causing thrombosis, and later cicatricial contraction, is not at all successful in these cases. In the patient mentioned above, numerous injections of alcohol were made without any success. This method is even dangerous, as thrombi may become separated and carried into the large dilated veins leaving the growth, causing fatal embolism.

Cauterization should be entirely discarded, as severe, even fatal hæmorrhage may follow separation of the eschar.

Amputation may be indicated when tumors of this character situated upon the extremities become ulcerated, leading to frequent and profuse hæmorrhages, and when phlegmons develop, as incisions cannot be made into these growths.

LYMPHANGIOMAS

Lymphangiomas are much more uncommon than hæmangiomas. They appear in three principal forms, between which are many transitional stages.

Lymphangioma Simplex.—The simple lymphangioma (lymphangioma simplex) corresponds to the simple hæmangioma, as it develops from a proliferation of the lymphatic vessels of a limited area of the skin and

subcutaneous tissues. The connective tissues lying between the newly formed vessels usually proliferate to form a part of the tumor. These tumors occur in the form of congenital, flat, or slightly nodular, but little circumscribed thickenings of the skin of the face and neck, and are to be regarded as true tumors. Transitional stages between the simple and cavernous forms are frequently found. Lymphangiectases developing after chronic inflammations and associated with the diffuse hyperplasias of connective tissues occurring in elephantiasis are to be differentiated from simple lymphangiomas (*vide* Figs. 240 and 241, pp. 649 and 651). Lymphangioma tuberosum cutaneum multiplex (Kaposi) occurring in the form of yellowish-brown nodules, never becoming larger than a lentil, which may be scattered throughout the skin of the entire body, also apparently belongs to the lymphangiectases.

Lentigines, Freckles, Flesh Warts, etc.—The endothelium lining the lymphatic vessels may proliferate, filling completely the lumina of the vessels. New growths develop in this way which should be classified with endotheliomas. Ziegler classifies a number of pathological changes occurring in the skin, such as *pigmented nævi, lentigines, freckles,* and *flesh warts,* commonly known as hypertrophic lymphangiomas, with endotheliomas. All these have this in common—they are composed of round collections or cordlike masses of proliferated endothelial cells of the lymphatic vessels lying in a connective-tissue reticulum. Borst believes that connective-tissue proliferation predominates in the pigmented nævi (therefore the term fibroma melanodes), while Soldan has shown that part at least of the pigmented nævi, like soft warts, are connected with the cutaneous nerves (*vide* p. 328).

Lymphangioma Cavernosum.—The lymphangioma cavernosum is the most common form of the diffuse, non-encapsulated lymphangiomas. It is of congenital origin, or develops in later life from congenital beginnings, and occurs in the skin, subcutaneous, and intermuscular connective tissues. Histologically it resembles quite closely the cavernous hæmangioma, as the tumor is composed of irregular spaces filled with lymph and communicating with each other. These spaces are lined by endothelium and are surrounded by a meshwork of fibrous tissue containing smooth muscle, elastic fibers, and small lymph nodes. A part of the lymphatic vessels of the normal surrounding structures communicate with the spongy tissue of the tumor, so that when pressure is made the tumor gradually decreases in size, and regains its normal size again when the pressure is removed.

Most Common Sites and Clinical Course.—Cavernous lymphangiomas occur most frequently in the cheeks (being occasionally bilateral), in the tongue, lips, eyelids, and lateral regions of the neck. The new growths may appear in the following different forms:

1. In the cheeks (macromelia, Fig. 332) and neck, as flat, loose swellings, covered by normal, yellowish, or reddish skin which cannot be raised from the tumor. The tumor gradually becomes continuous with the normal surrounding tissues.

2. As nodular, sometimes transparent, tumorlike thickenings of the lips (macrocheilia), concha (*vide* Fig. 333), and eyelids.

3. As unshapely enlargements of the tongue (macroglossia, Fig. 334), which becomes rigid and protrudes from the mouth, and as lobulated growths of considerable size (elephantiasis congenita lymphangiectatica) of the face and neck, the subcutaneous tissues also being involved. Encephaloceles and myeloceles not infrequently lie concealed beneath lymphangiomas, developing over the places where these malformations occur. Lymphangiomas of the mucous membranes of the pharynx and soft palate sometimes appear as soft, red elevations. These at times become inflamed, and as the inflammation is accompanied by

FIG. 332.—MACROMELIA CAUSED BY A CONGENITAL CAVERNOUS LYMPHANGIOMA.

FIG. 333.—CONGENITAL CAVERNOUS LYMPHANGIOMA OF THE EAR.

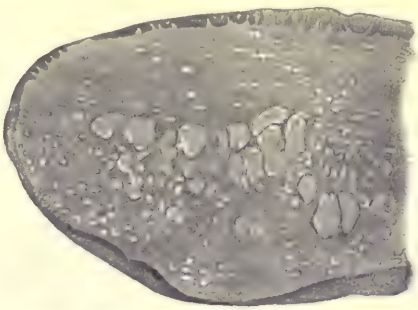


FIG. 334.—LYMPHANGIOMA OF THE TONGUE (MACROGLOSSIA), AS SEEN UNDER A MAGNIFYING GLASS.

fever, a diagnosis of diphtheria may be made, especially if, as in a case observed by Suckstorff, a membrane forms as a result of the coagulation of lymph discharged from the tumor.

The slow growth is continuous, but may be interrupted by rapid increase or decrease in size, due, especially in lymphangiomas of the face, to infection with bacteria entering from the mucous membranes (Tavel). This rapid increase and decrease in size is common to the clinical picture of all lymphangiomas. The cavernous tissue infiltrates the skin and sends out conelike processes between the muscles, displacing and surrounding nerves and blood vessels. Cases have been observed in which even bone has been destroyed by these growths (Katholicki).

Diagnosis.—The diagnosis of a cavernous lymphangioma may be based upon the poorly defined margins of the tumor, its soft, relaxed consistency, indistinct fluctuation, compressibility, varying in different parts of the tumor, and upon its being covered by skin which is not adherent and but little discolored.

Lymphangioma Cysticum.—This form of lymphangioma is much more frequently encapsulated than the cavernous, even the processes extending into adjacent tissues being surrounded by a capsule. The sharp boundaries are wanting only in the transitional forms which contain cavernous tissue. The cystic lymphangioma consists of single or multiple cysts, varying in size from a pinhead to a child's head. The cysts contain a serous fluid which, after injury, may be mixed with blood. The cysts do not communicate with neighboring lymphatics, and do not become smaller on pressure. Their inner surfaces are lined with endothelium, and the walls consist of thick fibrous tissue arranged in the form of a cavernous meshwork.

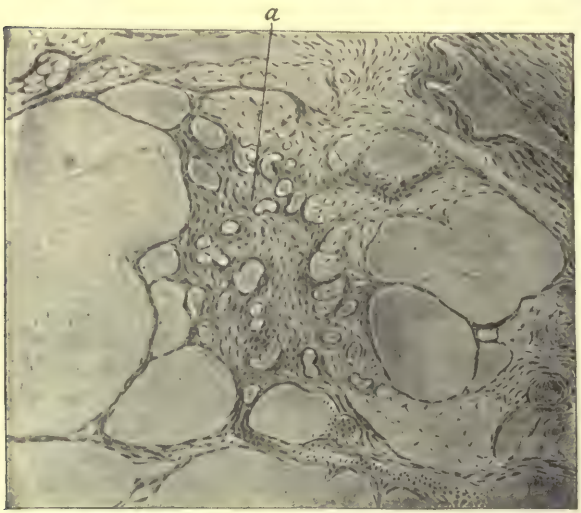


FIG. 335.—CYSTIC AND (*a*) CAVERNOUS LYMPHANGIOMA.

These tumors develop in the lateral regions of the neck, posterior to the sterno-cleido-mastoid, about the angle of the jaw, or in the supraclavicular fossa, and extend as they enlarge to the posterior part of the neck and toward the median line.

A large, cystic lymphangioma may extend from the jaw to the clavicle, from the median line in front to the same line posteriorly (Fig. 336). The new growths, depending upon the amount of fluid they contain, may be elastic and fluctuating, covered by skin which is tense but not adherent, or relaxed and soft. When the walls of the cysts are relaxed, irregularities produced by ledgelike projections and thickenings may be palpated. As they enlarge, cystic lymphangiomas may exert pressure upon the trachea, œsophagus, and large vessels, and become dangerous. This is especially so when they enlarge suddenly as the result of inflammation, which may end in suppuration.

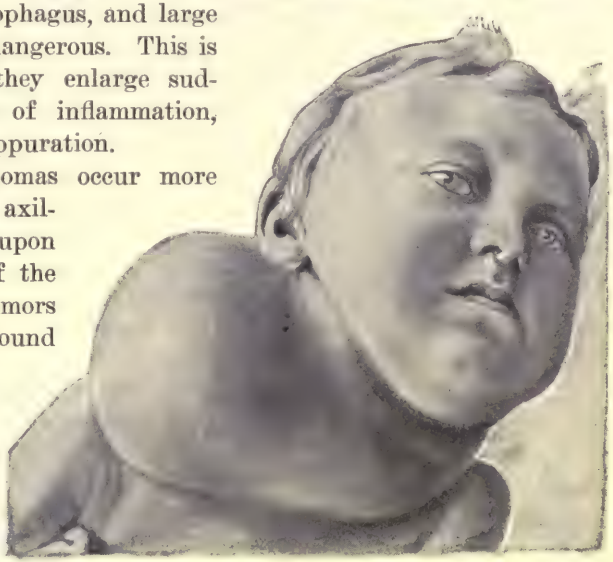


Fig. 336.—Congenital, Cystic Lymphangioma (Cystic Hygroma of the Neck). Cured by extirpation.

Cystic lymphangiomas occur more rarely in the cheeks, axillary fossa, groin, and upon the flexor surfaces of the extremities. These tumors are occasionally found upon the anterior surface of the sacrum and in the root of the mesentery. The latter contain chyle. The diagnosis of the nature of multilocular cysts, occurring where lymphangiomas are common, is not difficult. It is often extremely difficult to make an accurate diagnosis of the small unilocular forms, as one has also to consider branchial and blood cysts when they occur in the neck, echinococcus cysts and lipomas when they occur in the cheek.

Varicosities of the lymphatic vessels, developing in areas with a rich lymphatic supply, give rise to much the same clinical picture as do multilocular lymphangiomas. These tumors occurring about the sacrum, must be differentiated from dermoid cysts and teratoid tumors; at the root of the mesentery, from a number of different kinds of cysts.

Origin of Lymphangiomas.—Lymphangiomas are almost exclusively of congenital origin. They are the result of a disturbance in embryonic development, being frequently associated with other anomalies, such as myeloceles, encephaloceles, etc. Not only the lymphatic vessels, but also the fatty and fibrous tissues and the smooth musculature of the walls of the larger lymphatics, participate in these growths (Ribbert).

Changes Which May Occur in Lymphangiomas.—Lymphangiomas are benign growths, notwithstanding the fact that they frequently are not encapsulated and may be so situated or become so large that they threaten life. Inflammation may be followed by cicatricial contraction and spontaneous cure. It may also lead to the most serious consequences, such as exhausting lymph fistulæ or the extension of the inflammation along the lymphatic vessels communicating with the tumor, causing phlegmon of the orbit, mediastinitis, meningitis, etc., depending upon the position of the growth.

Indications for Treatment and Technic.—Encapsulated cystic tumors should be extirpated. If the skin is adherent to the tumor it should be removed at the same time. If one proceeds slowly and cautiously, frequently the entire tumor with all its processes may be removed by blunt dissection (sponges and tissue forceps) without rupturing it. Separation of adherent nerves offers the greatest difficulties. Cavernous portions of a tumor can never be completely removed.

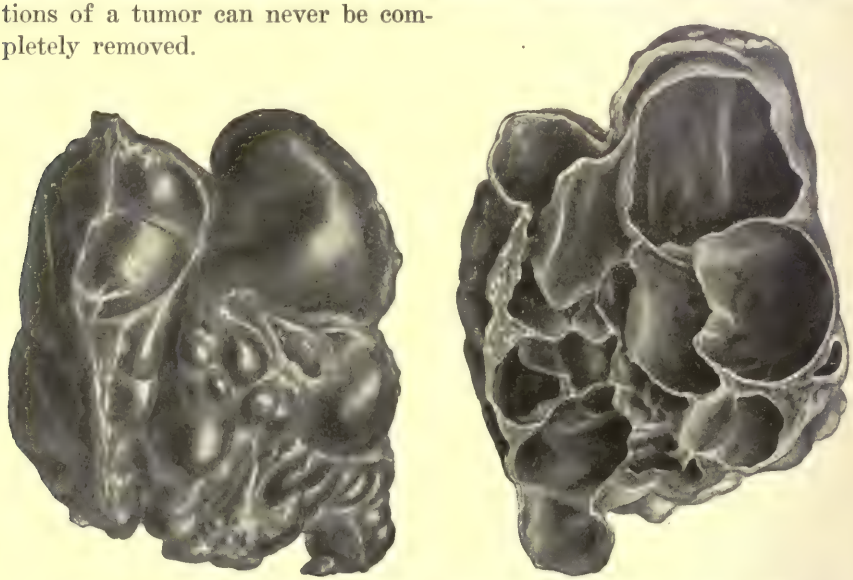


FIG. 337.—SPECIMEN REMOVED FROM PATIENT REPRESENTED IN FIG. 336.

After removal of the tumor the wound should be accurately sutured and a compression dressing applied, the accumulation of lymph and the development of lymph fistulæ being prevented in this way.

Incision of the cyst and tamponade, recommended by Wölfler, should not be employed, as Nasse has shown that there is danger of infection which may persist indefinitely if this method is employed.

The simple and cavernous lymphangiomas cannot be radically removed, and even partial removal by cuneiform excision, as recommended

in the treatment of macroglossia and macrocheilia, is not without danger, as lymph fistulæ and progressive inflammation may develop. In the treatment of these cases, especially in children, one must be content with less efficient but less dangerous methods, such as the injection of alcohol, tincture of iodin, and one per cent solution of zinc chlorid. If frequently repeated they cause at least a cicatricial contraction of parts of the tumor and a decrease in size. Cauterization should be discarded, as it has the same disadvantages as the incomplete operation.

LITERATURE.—*v. Bramann.* Ueber Chyluszysten des Mesenterium. Arch. f. klin. Chir., Bd. 35, 1887, p. 201.—*Engelbrecht.* Angioma arteriale racemosum. Arch. f. klin. Chir., Bd. 55, 1897, p. 347.—*Fr. Fischer.* Krankheiten der Lymphgefässe, Lymphdrüsen und Blutgefässe. Deutsch Chir., 1901, Rankenangiom, p. 222.—*Heine.* Ueber Angioma arteriale racemosum. Vierteljahrsschr. f. prakt. Heilkunde, 1869.—*Hildebrand.* Ueber multiple kavernöse Angiome. Deutsche Zeitschr. f. Chir., Bd. 30, 1889, p. 91.—*Honsell.* Ueber Alkoholinjektionen bei inoperablen Angiomen. Beitr. z. klin. Chir., Bd. 32, 1902, p. 251.—*Katholicki.* Ein Fall von Lymphangiom des Vorderarmes. Chir. Kongr.-Verhandl., 1903, I, p. 61 and Diskuss., p. 125, Payr, v. Bramann.—*A. Kruse.* Ueber das Chylangioma cavernosum. Virch. Arch., Bd. 125, 1891, p. 488.—*Küttner.* Ueber die intermittierende Entzünd. d. Lymphangiome. Beitr. z. klin. Chir., Bd. 18, 1897, p. 728.—*Laewen.* Ueber genuine diffuse Phlebarteriektasie a. d. ob. Extremität. Deutsche Zeitschr. f. Chir., Bd. 68, 1903, p. 364.—*Lieblein.* Ueber einen durch Alkoholinjektionen geheilten Fall von Angioma racemosum des Kopfes. Beitr. z. klin. Chir., Bd. 20, 1898, p. 27.—*E. Müller.* Zur Kasuistik der Lymphangiome. Beitr. z. klin. Chir., Bd. 1, 1885, p. 498.—*H. Müller.* Ein Fall von arteriellem Rankenangiom des Kopfes. Beitr. z. klin. Chir., Bd. 8, 1892, p. 79.—*W. Müller.* Zur Technik der Operation grösserer Hämangiome und Lymphangiome. Beitr. z. klin. Chir., Bd. 37, 1903, p. 565.—*Muskatello.* Ueber das primäre Angiom der willkürlichen Muskeln. Virch. Arch., Bd. 135, 1894, p. 277.—*Narath.* Ueber retroperitoneale Lymphzysten. Chir. Kongr.-Verhandl., 1895, II, p. 396.—*Nasse.* Ueber Lymphangiome. Arch. f. klin. Chir., Bd. 38, 1889, p. 614.—*Payr.* Ueber Verwendung von Magnesium zur Behandlung von Blutgefässerkrankungen. Deutsche Zeitschr. f. Chir., Bd. 63, 1901, p. 503.—*Ranke.* Zur Anatomie der serösen Wangenzysten. Arch. f. klin. Chir., Bd. 22, 1878, p. 707.—*Ribbert.* Wachstum und Genese der Angiome. Virch. Arch., Bd. 151, 1898, p. 381.—*Riethus.* Ueber primäre Muskelangiome. Beitr. z. klin. Chir., Bd. 42, 1904, p. 454.—*Ritschl.* Ueber Lymphangiome der quergestreiften Muskeln. Beitr. z. klin. Chir., Bd. 15, 1896, p. 99.—*Rottgans.* Aneurysma cirsoides. In Hildebrand's Jahresber., 1897, p. 346.—*Sachs.* Die von den Lymphgefässen ausgehenden Neubildungen am Auge. Ziegler's Beitr. z. pathol. Anat., Bd. 5, 1889, p. 99.—*Samter.* Ueber Lymphangiome der Mundhöhle. Arch. f. klin. Chir., Bd. 41, 1891, p. 829.—*Strauch.* Intramuskuläres kavernöses Angiom (Masseter). Deutsche Zeitschr. f. Chir., Bd. 62, 1902, p. 323.—*Suckstorff.* Lymphangiom des Rachens mit Bildung kruppöser Membranen. Beitr. z. klin. Chir., Bd. 27, 1900, p. 185.—*Sutter.* Beitr. zu der Frage von den primären Muskelangiomen. Deutsche Zeitschr. f. Chir., Bd. 76, 1905, p. 368.—*Tavel.* Ueber die schubweise auftretende entzündliche Schwellung der Lymphangiome. Zentralbl. f. Chir., 1899, p. 817.—*Weichselbaum.* Chylangioma cavernosum des Mesenteriums. Virch. Arch., Bd. 64, 1875, p. 145.—*Wegner.* Ueber Lymphangiome. Arch. f. klin. Chir., Bd. 20, 1877, p. 641.

CHAPTER VI

SARCOMAS

Definition and Nature.—Sarcomas (from the Greek σάρξ, meaning flesh) are malignant tumors which are derived from mesoblastic tissues. They are composed of immature, unripe elements, while the benign connective-tissue tumors, such as the fibromas, chondromas, and osteomas described in the preceding chapters, are composed of mature, fully developed elements. In sarcomas the cellular elements predominate over the intercellular substance, the former proliferating rapidly and without restraint. Sarcomas are closely related to embryonal tissues and to granulation tissue developing in wounds and in chronic inflammatory processes (especially the infectious granulomas). The cells composing the latter, however, complete their cycle of development forming adult connective tissues if favorable conditions are provided, while the cells composing a sarcoma maintain their embryonal characteristics. They have lost their ability to form tissues of an adult type (Borst) and proliferate without restraint at the expense of the organism. These tumors form the malignant group of the connective-tissue tumors corresponding to carcinomas, malignant tumors arising in epithelial tissue.

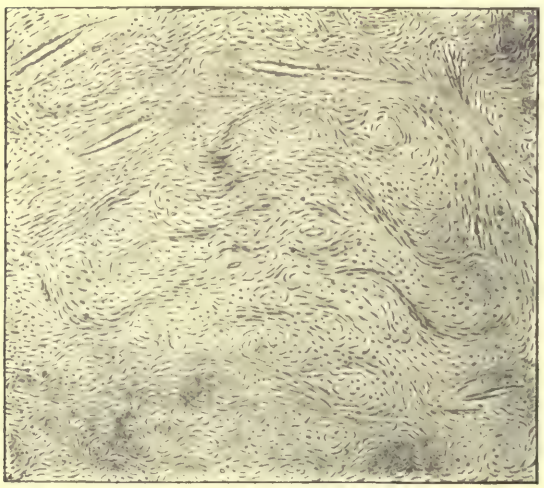


FIG. 338.—FIBROSARCOMA.

Classification.—Sarcomas differ in histological characteristics and in their clinical courses. Frequently it is impossible to determine the clinical peculiarities of a sarcoma by histological findings; therefore it is difficult to make a classification in which both the histological picture and the clinical course are taken into consideration. Ribbert's classification seems to be the best. He distinguishes between sarcomas composed:

1. Of cells of any of the connective tissues;
2. Of cells resembling lymph corpuscles;
3. Of mucoid tissues;
4. Of pigment cells.

Characteristics Common to all Sarcomas.—There are certain characteristics common to all sarcomas which may be more marked in some than in others.

Sarcomas consist principally of cellular elements, the intercellular tissue, if present, being greatly reduced in amount. The intercellular substance may be fibrillar, cartilaginous, bony, or mucoid, depending upon the origin of the tumor. Remnants of the infiltrated and degenerating tissues may also be found between the cells. Blood vessels, the development of which varies in different tumors, form the third constituent part of sarcomas. As in granulation tissue, the newly formed capillaries form the framework for the proliferating groups and columns of cells. Among the thin-walled vessels, arteries and veins cannot be differentiated from each other. The vessels consist of spaces or clefts in the tumor tissues. In some cases the endothelium lining the vessels rests directly upon the tumor cells, while in other cases the connective-tissue stroma of the tumor forms a fairly well-defined wall. If these spaces, surrounded by stroma, become filled with cells, structures histologically resembling alveoli (alveolar sarcoma) are produced.

Fig. 339.—Large Spindle-Cell Sarcoma.

If the vessels are numerous and the proliferation of the tissues immediately adjacent to them is marked, transitional stages to the peritheliomas, in which the proliferation begins in the perivascular endothelial cells, are found. This is especially true if the columns of cells surrounding the vessels do not become fused with each other, but remain separated by intercellular substance, lymph, or blood. These tumors are called angiosarcomas or, better, telangiectatic or cavernous sarcomas (Borst), as these terms can also be used for peritheliomas.

Mode of Growth.—The growth is usually expansive in the beginning, the adjacent displaced tissues forming a thin capsule. If operated upon at this stage, the tumor can sometimes be easily enucleated. Many sarcomas from the first, and all later on, infiltrate the surrounding tissue, replacing the normal structures. The increase in size of these tumors is due to the proliferation of the cells composing them, and not

to the transformation of surrounding elements into tumor tissue. Proliferation of the surrounding tissues is indicative of reactive growth and not of transformation into tumor tissue. The infiltrated tissues gradually undergo pressure atrophy. Pressure upon the larger vessels may cause necrosis. Even bone is disintegrated by the tumor cells. Cartilage resists their growth for a long time.

Regressive Changes.—A number of regressive changes, such as fatty and parenchymatous degeneration of the cells, necrosis of large areas, hæmorrhages, thrombosis of the large vessels, hyaline degeneration and obliteration of the blood vessels and death of the tissues surrounding them, may occur in any sarcoma. Softened areas and blood cysts then develop, and upon section yellowish, gelatinous, and hæmorrhagic foci may be seen upon a surface which, in the beginning, is of a grayish or red color throughout. Necrosis of the infiltrated skin leads to the formation of deep ulcers in which putrefactive inflammation may easily develop. It is interesting to note that these tumors occasionally become smaller following an attack of erysipelas (Busch), the cells undergoing a fatty degeneration and being absorbed (Spronck, Borst). X-rays have a similar effect upon the tumor cells.

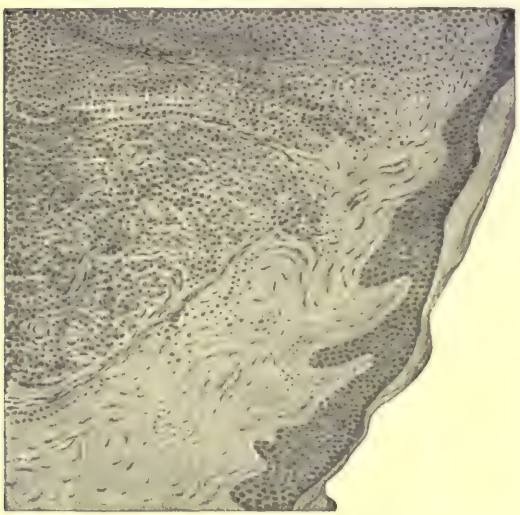


Fig. 340.—Round-Cell Sarcoma of the Skin.

Metastases.—The malignancy of sarcomas is indicated especially by the formation of metastases. The tendency to the formation of metastatic growths is least marked in the encapsulated tumors and in the relatively highly developed fibrosarcomas; it is most marked in the cellular, rapidly growing, infiltrating forms, especially in the round-cell sarcomas. Secondary nodules may develop by way of the lymph stream in tissues adjacent to the tumor or in regional lymph nodes. Metastases by way of the lymphatics are not, however, the rule as in carcinoma. In sarcomas metastases by way of the blood stream are more common, as the cells invade the vessels of the surrounding tissues or those of the tumor itself. A progressive, intravascular growth then develops, or, as more frequently happens, a small group of

cells or single cells are separated and carried away in the blood stream to be deposited in the lungs, liver, spleen, bone marrow, the kidneys, and in other viscera and tissues. The metastatic growths repeat the structure of the primary tumor, but proliferate still more rapidly.

Fever and Anæmia Associated with Sarcomas.—The effect of the sarcoma upon the organism is often indicated by an irregular fever, due to the absorption of pyrogenic substances from the tumor (hæmorrhages, products of decomposition), and by a progressive anæmia, which always suggests the formation of general metastatic growths.

Age at Which Sarcoma Develops.—Sarcomas occur most commonly in middle-aged people, more rarely in the young and old. Congenital sarcomas are relatively common. It is striking that they are most common in powerful, healthy men. As a rule, the primary tumor is single, but primary multiple tumors have been observed.

Cause of Sarcoma Formation.—The essential cause of sarcoma formation has not been determined. There are a number of objections which may be raised against the parasitic theory. There are a number of facts which seem to justify the theory that these tumors arise from rests, displaced during embryonal or later life, such as: their congenital occurrence; the presence of sarcoma tissue in (teratoid) mixed tumors, which certainly develop from tissue displaced during embryonal life; the development of tumors from displaced adrenal rests, from undescended and displaced testicles, and from congenital tumors, such as neurofibromas and soft warts; likewise, the development of bone and cartilage in sarcomas of the soft tissues and

FIG. 341.—SOFT, VASCULAR SARCOMA OF THE LEFT HALF OF THE FACE, DEVELOPING APPARENTLY FROM THE BULB OF THE EYE. The growth has extended into the nose, pharynx and mouth.

the occasional development of sarcoma tissue in any of the benign connective-tissue tumors. Germinal tissue apparently may be displaced in later and as well as in embryonal life, during growth, in inflammatory and regenerative processes (Ribbert).

A sarcoma developing in a callus following a fracture may be cited as an example of a tumor developing from tissues displaced during regenerative processes. According to clinical experience, it is highly probable that the trauma is only the exciting cause in such a case,

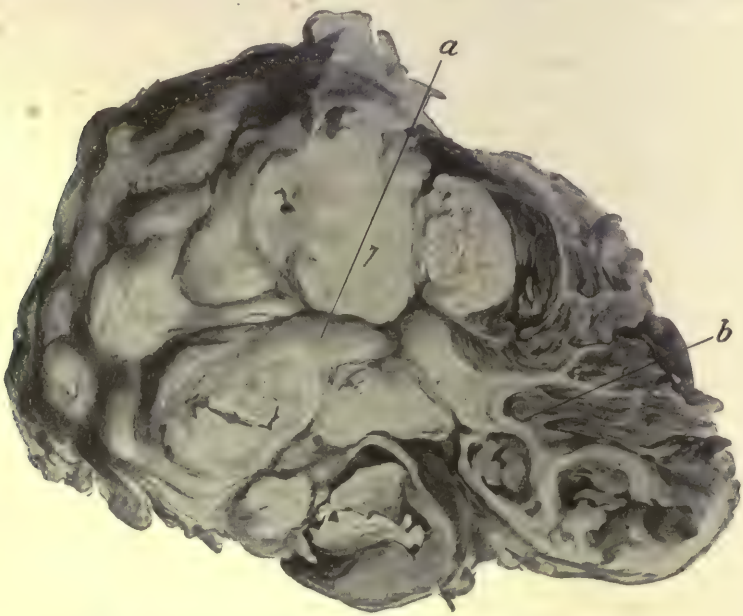


FIG. 342.—SARCOMA TISSUE (*a*) WHICH HAS INVADED MUSCLE (*b*).

stimulating a tumor to more rapid growth, which has previously existed, but has given rise to no symptoms. In the example cited above it is more probable that a tumor existed before the fracture than that a tumor developed from the callus.

(a) SARCOMAS DEVELOPING FROM THE DIFFERENT CONNECTIVE TISSUES

Sarcomas, which develop from any of the connective tissues, may contain but little interstitial substance and resemble closely histologically embryonal connective tissues. Some of these tumors contain cartilage and bone, indicating that they have developed from the skeleton (chondro- and osteosarcoma).

FIBROSARCOMAS

Sarcomas arising from fibrous tissues are most commonly composed of spindle cells (sarcoma fusocellulare), less frequently of round cells (sarcoma globocellulare), small and large cells with many transitional forms being found. Besides the soft, medullary forms, which usually are very malignant, there are the firm, less malignant forms containing relatively large amounts of intercellular tissue. The differences in consistency and appearance depend usually upon the number of blood vessels, the amount of blood pigment, upon hæmorrhages and regressive changes. Sometimes the cut surface has a homogeneous, grayish red or dark red appearance; at other times it is dotted with hæmorrhagic foci and cysts.

FIG. 343.—ROUND-CELL SARCOMA OF THE CHEEK.

Histology of Fibrosarcoma.—The spindle cells have nuclei centrally situated, and each end of the cells is provided with a long process. The large cells are, as a rule, irregular in shape, being round or fusiform, oval or serrated. Often they are arranged in a fasciculated manner. The intercellular fibrillæ are most abundant in the fibrosarcomas, which can only be differentiated from fibromas by their cells of unequal size, rich in cytoplasm and containing large nuclei.

Spindle-cell sarcomas usually are firm, hard tumors. They are less malignant than round-cell sarcomas, causing less local disturbance, growing less rapidly, and forming metastases, which are less extensive, later. The spindle-cell sarcoma composed of large cells are, however, almost as malignant as the round-cell varieties.

Round-cell sarcomas are composed either of small, round cells with little cytoplasm, or of large epitheliallike cells, rich in protoplasm and containing vesicular nuclei. If the cells lie imbedded in a well-developed

stroma (alveolar sarcoma) the tumor may resemble a carcinoma very closely histologically. Round-cell sarcomas are characterized by a rapid, infiltrating growth. They are more frequently soft than hard.

Usually a single type of cell is found only in spindle-cell sarcomas. In other forms of sarcoma, while one type of cell predominates, a number of other types are also found. Irregular, atypical, mitotic figures and multinuclear cells, the cytoplasm of which is not capable of division, are indicative of defective processes of growth.

FIG. 344.—SARCOMA OF THE SKIN OF THE THIGH IN A WOMAN SIXTY YEARS OF AGE (SMALL-CELL VARIETY).

Giant-cells, which resemble closely osteoclasts, are found in addition to the round and spindle cells in fibrosarcomas, especially in those developing from bone. The giant-cell sarcomas, composed principally of spindle cells, develop from the periosteum of the jaw (epulis) or from the marrow of long, hollow bones (*vide* p. 853). They form a relatively benign group of tumors.

Clinical Course.—The clinical course of the sarcomas arising from fibrous tissue depends upon the position of the tumor and its degree of

malignancy. The diagnosis is often difficult, especially when the tumor is just beginning to develop.

Sarcomas of the Skin.—Sarcomas of the skin appear as rapidly growing, round, well-defined nodules and masses, or as pedunculated, fungiform, and nodular tumors, varying in consistency. They have a bluish-red color, as they contain a large number of vessels, and, in the beginning at least, are covered by epidermis. Later the skin becomes infiltrated, and large, deep ulcers may form, or the surface of the tumor, which bleeds easily and profusely, may be covered by crusts. Sarcomas of the skin often develop from warts and papillomas. Frequently they are multiple from the beginning. So long as they are not large and do not extend deeply, they move with the skin. Rapid growth indicates malignancy. The nodular surface of the tumor, or the fissured, irregular ulcer may resemble

Fig. 345.—Fibrosarcoma of the Aponeurosis of the Occipito-Frontalis (Man Fifty Years of Age).

Fig. 346.—Multiple Sarcomas of the Skin in a Man Forty Years of Age (Small-Cell Variety).

closely the changes found in carcinoma. The absence of indurated lymphatic enlargements enables one to exclude the latter. It is often difficult to distinguish the multiple sarcomas from mycosis fungoides, a peculiar disease, the etiology and exact nature of which are unknown.

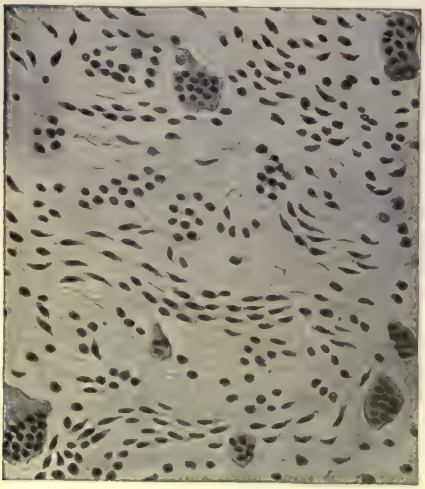


FIG. 347.—FIBROSARCOMA WITH GIANT-CELLS (EPULIS).

Sarcomas of Subcutaneous Tissues.—Sarcomas developing in the subcutaneous tissues appear as round nodules with slightly uneven, tuberculated surfaces. In the beginning they are sharply defined against the surrounding tissues, but later infiltrate them, become adherent to the skin, and ulcerate through it. They may develop from fibromas of the nerves. The diagnosis is based principally upon the rapid growth. They may be easily differentiated from fibromas, but with difficulty from rapidly developing subcutaneous gummas. A positive diagnosis at times can be made only by examining pieces of tissue, or after ulceration, when the products characteristic of syphilitic lesions are wanting.

FIG. 348.—VERY VASCULAR SARCOMA OF THE SKIN. The tumor has broken through the skin which surrounds it like a collar.

FIG. 349.—ROUND-CELL SARCOMA OF THE SKIN.

Sarcomas of Mucous Membrane.—Sarcomas of the mucous membrane may be composed of either round or spindle cells. They usually grow rapidly. They appear as nodular tumors with broad bases, more rarely as pedunculated or poorly defined thickenings, varying in consistency. After destruction of the mucous membrane, which is apt to occur early, craterlike ulcers form. They develop from the submucosa, more rarely from the intermuscular and submucous connective tissues, and occur in the stomach, intestines, and sometimes in the tongue and trachea. Sarcomas of the mucous membranes are much less frequent than are carcinomas, with which they have many clinical symptoms in common.

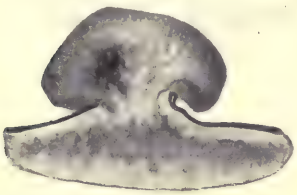


FIG. 350.—SECTION OF A PEDUNCULATED SARCOMA OF THE SKIN (VASCULAR SPINDLE-CELL SARCOMA) OF THE FINGER. The cutis does not extend beyond the pedicle.

FIG. 351.—ULCERATED ROUND-CELL SARCOMA OF THE STOMACH (WOMAN TWENTY-THREE YEARS OF AGE. RESECTION OF RIGHT HALF OF STOMACH, RECOVERY).

Intermuscular Sarcomas.—Intermuscular sarcomas are most commonly composed of large cells and grow rapidly. They may form enormous tumors entirely surrounding the bones of the part involved. The diagnosis is often difficult when the tumor is seen in its early stages, as one has to exclude a gumma developing in the muscle. The diagnosis may be difficult even in the advanced stages, as it may be impossible to determine whether the tumor has developed from soft tissues or bone. Besides the very cellular, soft, malignant tumors, there are also hard, fibrous, intermuscular tumors which grow slowly and are partially encapsulated.

Both of these forms are also represented in sarcomas developing from fascia and the connective-tissue sheaths of blood vessels. The latter especially give rise to early symptoms by pressure upon nerves and blood vessels.

Sarcomas of the Periosteum.—The sarcomas developing from the periosteum—in rare cases they are multiple (Nasse)—are fibrosarcomas or

Fig. 352.—Small Round-Cell Sarcoma of the Hand, Developing from the Fascia. Amputation was soon followed by symptoms indicating involvement of the lungs. A piece of tissue was expectorated, which resembled histologically the primary tumor. (From Prof. Bevan's Surgical Clinic.)

spindle-cell sarcomas, through which are distributed giant cells. They are common, appearing as nodular, hard tumors upon the alveolar processes of the jaws. A tumor of this character may be attached by a broad base or a rather narrow pedicle. Such a tumor is called an epulis. It is often difficult to differentiate between these tumors and fibromas which occur upon the jaws. These firm periosteal sarcomas also occur on other bones, especially on the ends of long, hollow bones. Spindle-cell sarcomas also occur in the dura, forming a part of the tumors known as fungus duræ matris. The fibrosarcomas developing from bone marrow contain a large number of giant cells and are relatively benign. Their position and clinical symptoms correspond to those of myeloid sarcomas.

The nodular, encapsulated tumors developing from the synovial sheaths of the flexor tendons of the fingers have a similar structure, and are, likewise, not very malignant. Their red or yellowish-brown color is due to the deposition of blood pigment following hæmorrhages caused by trauma.

Sarcomas of Nerves.—If a sarcoma develops from the connective tissues of a nerve, a nodular, fusiform thickening or a tumor which is encapsulated at first forms upon the nerves. These tumors grow more rapidly than fibromas. They press upon the nerves, giving rise to considerable pain, and may finally infiltrate the surrounding soft tissues. Occasionally a sarcoma develops from a simple fibroma which may have existed for some time.

Fig. 353.—Soft, Small, Spindle-Cell Sarcoma of the Fascia Lata.

Retroperitoneal Sarcomas. — Sarcomas of this group develop in the posterior abdominal wall and in the mesentery, forming large growths with nodular surfaces. They form much more extensive growths and develop more rapidly than retroperitoneal fibromas.

Fibrosarcomas of the different organs (mammary, thyroid, and parotid glands, testicle, kidney, uterus, etc.) rapidly destroy the parenchyma of the organ involved. The serous membranes (pleura, peritoneum) are but rarely primarily involved.

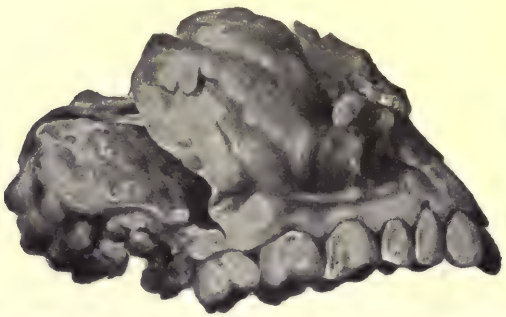


Fig. 354.—Central Giant-Cell Sarcoma of the Upper Jaw (Resection Preparation).

The different forms of primary sarcoma of the lymph nodes, excepting the lymphosarcomas, are exceptionally rare.

Diagnosis.—Usually one must be content with making a general diagnosis of sarcoma, differentiating it from other forms of malignant

growths. Sometimes the diagnosis as to the histological structure of the tumor can be based upon the position of the tumor; for example, periosteal giant-cell sarcomas are common upon the alveolar processes of the jaws. Often it is impossible to differentiate between a lymphosarcoma, a chondrosarcoma, and a myxosarcoma. The color of a melanosarcoma, the consistency and position of an osteosarcoma enable one, as a rule, to make a positive diagnosis as to the character of the tumor.

FIG. 355.—GIANT-CELL SARCOMA OF THE MAMMARY GLAND (MOSTLY LARGE CELLS).

It is often exceedingly difficult to make a diagnosis between a sarcoma and a deep inflammatory mass, especially a gumma. In these cases an exploratory incision is a much more rapid diagnostic method than antisyphilitic treatment.

Treatment.—The treatment of a sarcoma consists of as early and complete removal as possible, if there are no demonstrable metastatic growths. If the tumor is so situated that an amputation is possible, it should be performed if there are no contra-indications.

CHONDRO- AND OSTEOSARCOMAS

Chondro- and osteosarcomas develop from the skeleton; more rarely from the soft tissues.

Chondrosarcomas are closely related genetically to chondromas, from which they may develop, and occur in the same regions. They are characterized, like the chondromas, by the formation of a hyaline ground substance. They differ from the chondroma in that they are more cellular, being composed of groups or columns of round, fusiform, or polymorphous cells, surrounded by small or large islands of cartilaginous substance. Within this ground substance lie isolated groups of cartilage cells, usually without a capsule.

Appearance of Cut Surface.—The cut surface of a chondrosarcoma does not have the homogeneous appearance of the cut surface of the chondroma. The cartilaginous parts appear as opal, bluish areas in the soft, reddish, sarcomatous tissue. Calcification of the cartilage is indicated by the development of white areas in the tumor, ossification by its hardness (chondro-osteosarcomas). Softening with subsequent liquefaction may lead to the formation of large cystic cavities. Chondrosarcomas grow rapidly, often attaining enormous size, infiltrating the soft tissues, and destroying bone. Not infrequently they form metastases, as the proliferation of the cells is so rapid that there is scarcely time for the formation of a capsule.

Osteosarcomas. — Osteosarcomas, or ossifying sarcomas, are much more common than chondrosarcomas. Strictly speaking, one means by an osteosarcoma a tumor which contains bone or its antecedents, and not a fibrosarcoma developing from bone. It is not always possible to make a sharp distinction, as a fibrosarcoma developing from the periosteum may contain bone formed by the reactive growth of the latter.

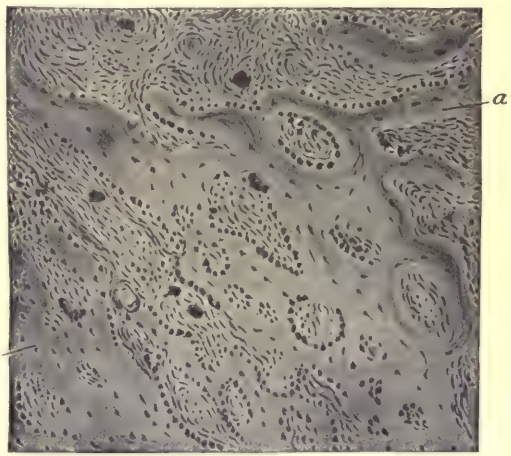


FIG. 356.—PERIOSTEAL OSTEOSARCOMA OF THE ULNA. Sarcomatous tissue with giant-cells and osteoblasts may be seen between newly formed calcified (*a*) and osteoid (*b*) lamellæ.

Osteosarcomas develop most frequently in the ends of long hollow bones, in the bones of the pelvis, the scapula, the clavicle, the sternum, the bones of the skull, the short, hollow bones, the vertebræ, the os calcis, and the patella. They

develop most frequently during the period of growth and are divided into periosteal (peripheral) and myelogenous (central) tumors.

Periosteal sarcomas (Fig. 356) are composed mostly of spindle cells, but also contain round- and giant-cells. The cells lie in a stroma, which resembles bone, and sometimes is arranged in regular lamellæ, and at other times has no definite arrangement whatever. If the tumor contains only delicate, non-calcified, osteoid lamellæ, it is spoken of as an osteoid sarcoma. These tumors are closely related to those less malignant tumors which are composed of broad, non-calcified lamellæ of a cartilaginous ground substance, between which are found irregular cells, which have been called *osteoid chondromas* by Virchow. The formation of bone is rarely evenly distributed throughout the whole of the tumor. It is much more advanced in the older, central parts of the tumor than in the cellular, and therefore softer, peripheral parts. Areas composed of tissue resembling bone alternate with those composed of osteoid or chondroid tissue. The lamellæ, which frequently contain and are surrounded by osteoblasts, either form a spongy framework in which the sarcoma cells and the vessels supplying the marrow lie, or are arranged so that after maceration (Fig. 357) they appear as needles and projections radiating in all directions or irregularly grouped.

FIG. 357.—PERIOSTEAL OSTEOSARCOMA OF THE LOWER END OF THE FEMUR WITH A RADIATING ARRANGEMENT OF THE TRABECULÆ OF BONE.

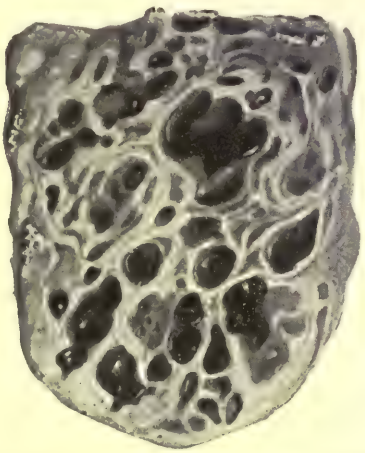


FIG. 358.—CYSTIC O TEOSARCOMA OF THE STERNUM.

Periosteal sarcomas appear in the beginning as circumscribed nodules, varying in consistency and covered by a layer of periosteum. They are most common in long, hollow bones, beginning as a rule about

the metaphysis. During later growth such a tumor may involve the entire bone, which then becomes transformed into a shapeless, nodular mass resembling a club. If the tumor grows slowly, forming considerable bone, only the superficial part of the cortex is destroyed and the base of the tumor is surrounded by osteophytes. If the tumor grows rapidly and perforates its capsule, it invades muscles, tendons, and

FIG. 359.—MYELOGENOUS SARCOMA OF THE LOWER END OF THE FEMUR, WHICH HAS EXTENDED ALONG THE SURFACE OF THE BONE AFTER RUPTURE THROUGH THE EPIPHYSIS.

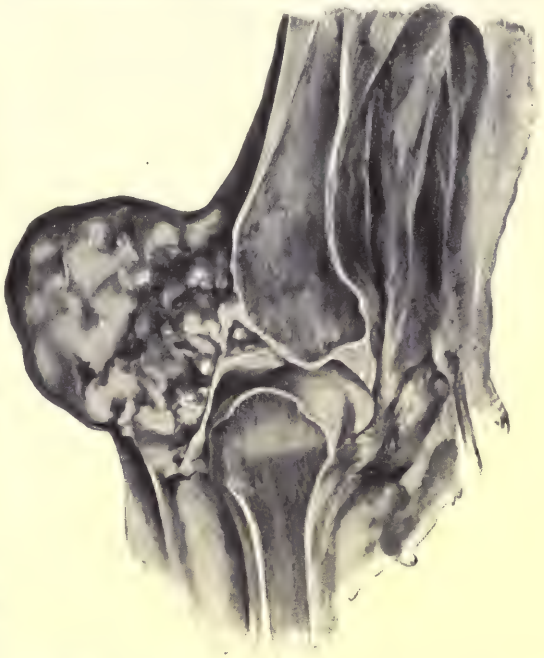


FIG. 360.—OSTEOID SARCOMA OF THE PATELLA (SAGITTAL SECTION THROUGH THE KNEE JOINT).

ligaments, and passes along the Haversian canals to the medullary cavity, destroying the cortex. The tumor can be differentiated from the bone marrow by its lighter color. In advanced cases, therefore, it is often difficult to determine whether the tumor originated in the periosteum or bone marrow. The articular cartilage resists for the longest time the invasion of the tumor, but the joint capsule may be early involved at its line of attachment to the bone, and the tumor may extend to the joint cavity in this way.

The clinical picture of the periosteal sarcoma resembles very closely that of the myelogenous. The periosteal has no bony shell surrounding it, which is present in the beginning of the myelogenous forms.

Myeloid Sarcomas.—The myelogenous osteosarcomas, or myeloid sarcomas, occur most frequently in the spongy ends of long, hollow bones

(especially in young people), being situated in the metaphysis close to the epiphyseal cartilage more frequently than in the epiphysis. They may, however, develop in any other of the bones. The mandible, carpal, and tarsal bones, the bones of the skull, the vertebræ, and pelvic bones are most frequently involved after the long bones. Multiple myeloid sarcomas have been observed, especially in association with osteitis deformans (p. 749).

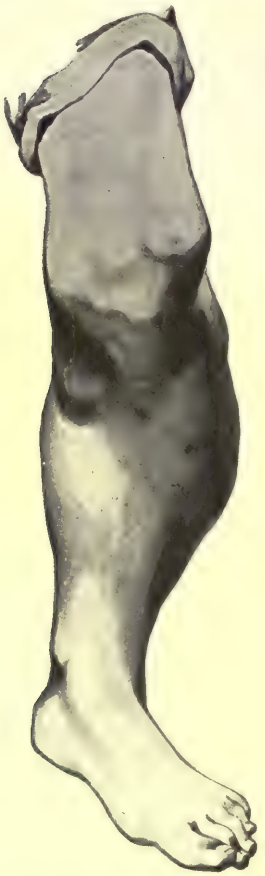


Fig. 361. — Osteosarcoma of the Fibula.

Histology.—They are composed of round and spindle cells or large cells of different shapes, and contain giant cells more frequently than the periosteal forms. Generally the formation of bone is less marked than the cellular proliferation. These tumors are exceedingly vascular and may pulsate. They are often spoken of as *bone aneurysms*. Thrills may be elicited upon palpation and bruits upon auscultation. Hæmorrhages within the tumor tissue are relatively common, and pigment is deposited in the tissues which assume a yellowish or reddish brown color. Softening with subsequent liquefaction leads to the formation of cysts, the walls of which contain trabeculæ of bone.

Mode of Growth.—Generally these tumors have an expansive growth for some time, and are therefore relatively benign, being the least malignant of all sarcomas. This is especially true of the tumors composed of spindle cells with a fibrous or osteal ground substance, and containing large numbers of giant cells. As the tumor grows the bone becomes expanded, but not, as in inflammatory processes, as the result of a thickening of the cortex. The cortex gradually undergoes a pressure atrophy from within as the tumor enlarges, and is not replaced by new bone formed from the periosteum. The bone surrounding a central tumor is gradually destroyed, and spontaneous or pathological fractures may occur. Some-

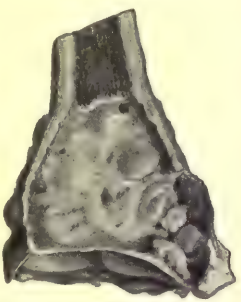


Fig. 362.—Myelogenous Giant-Cell Sarcoma of the Lower End of the Radius (Woman Thirty-five Years of Age. Resection Preparation. No Recurrence After Four Years).

times, especially in sarcomas of the jaw, the thin, yielding shell of bone imparts a "parchmentlike crackle" (Dupuytren) to the palpating finger. If the periosteal bone formation is not as rapid as the bone absorption, the bony capsule surrounding the tumor is sooner or later ruptured, and the tumor then extends to the soft tissues and invades the joint and surrounding bone. Infiltrating growth then predominates, resulting in the rapid formation of metastases.

Malignant Transformation.—The transition from a relatively benign to a malignant stage does not progress with the same rapidity in all cases. The soft, cellular forms (the so-called medullary forms) rupture through their capsule and assume an infiltrating growth much earlier than the firm forms (containing large numbers of giant-cells), which, even after extension to the soft tissues, may preserve for a long time their tendency to limitation and expansive growth.

FIG. 363.—CENTRAL OSTEOSARCOMA OF THE FEMUR WITH RUPTURE INTO THE SOFT TISSUES AND KNEE JOINT.

Symptoms.—Usually the first symptoms are pain and rapidly developing enlargement of the bone involved. In myeloid sarcomas of the bones of the extremities, spontaneous fractures and a serous exudate into the neighboring joint may develop early.

If the pain and enlargement of the bone have not been marked, it is often difficult to interpret correctly the clinical significance of the fracture or of the accumulation of fluid in the joint.

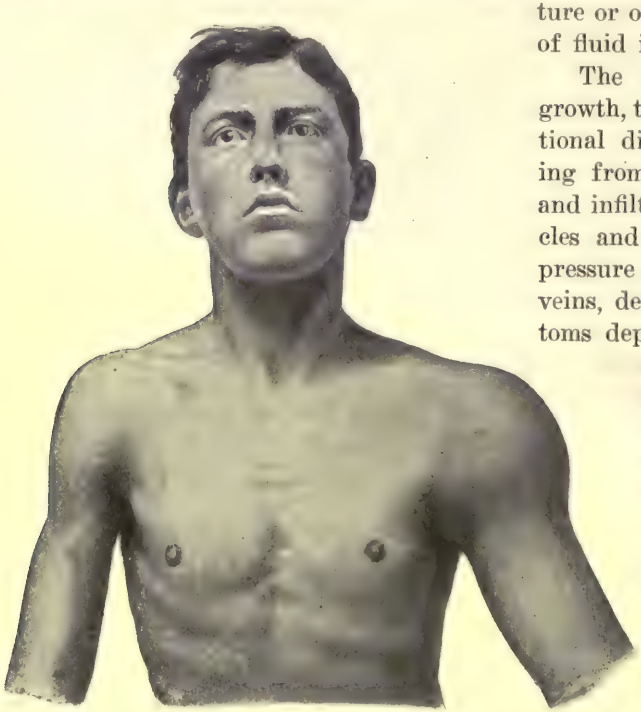


Fig. 364.—Osteosarcoma of the Upper End of the Humerus in a Patient Nineteen Years of Age.

The more rapid the growth, the earlier the functional disturbances, resulting from the displacement and infiltration of the muscles and nerves and from pressure upon the large veins, develop. The symptoms depend upon the position of the tumor (for example, a tumor growing into the cranial cavity from the surrounding bones produces symptoms of cerebral compression; a tumor developing from the bones of the thorax, symptoms of lung compression). Deep ulcers, which bleed profusely and easily become infected, follow infiltration of the skin.

Diagnosis.—The diagnosis in advanced cases is based upon the presence of a large, rapidly growing tumor which is firmly attached to the bone involved by a broad base. The boundaries of the tumor cannot be sharply defined, as in the advanced cases the tumor has already contracted adhesions with the surrounding muscles. Sometimes it can be demonstrated that the function of the muscle is interfered with or lost, indicating that it has been infiltrated by tumor tissue. In the large and rapidly growing tumors, symptoms of metastatic lung foci, accompanied by those of a pleuritic effusion, are frequently present.

It is often impossible to determine by the position and consistency of the tumor whether it developed primarily from the periosteum or medulla, or even from the soft tissues, whether it is an osteo-, chondro-, or fibrosarcoma. This, however, is of little practical importance. Myelogenous are more common than periosteal forms, and often cause spon-

taneous fractures. The early diagnosis is difficult. The symmetrical enlargement of the bone, accompanied by an acute hydrops of the neighboring joint, may suggest some chronic inflammatory process, such as tuberculosis or syphilis. Sarcomas frequently develop in parts of the bone where chronic inflammatory processes (such as tuberculosis and syphilis) are common, and are often accompanied by some fever. The small periosteal sarcomas resemble quite closely fibromas, chondromas, and osteomas, being nodular and hard. A spontaneous fracture favors the diagnosis of sarcoma, but even when a spontaneous fracture is present a positive diagnosis cannot always be made. A sarcoma cannot be excluded even when the lesions are multiple, but the probabilities are that when lesions are primarily multiple the tumors are not malignant.

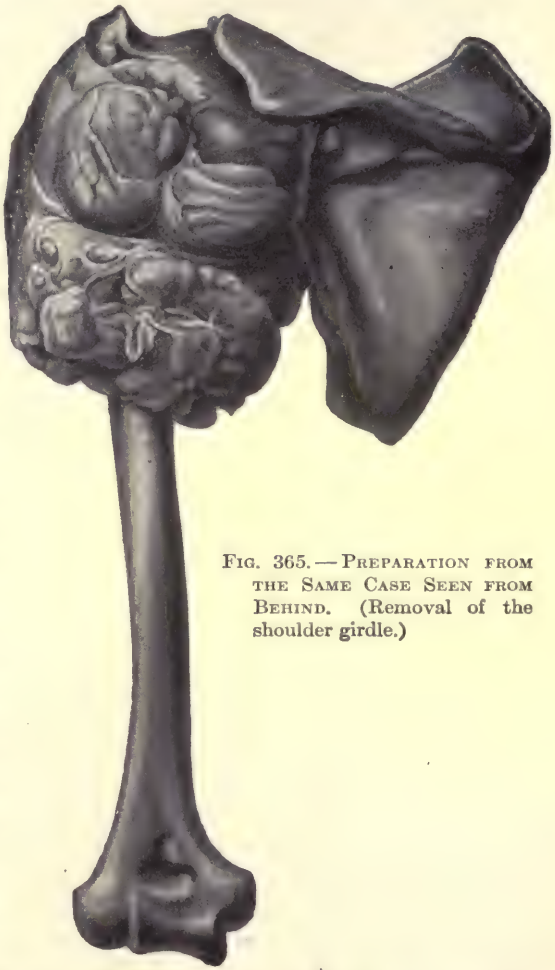


FIG. 365. — PREPARATION FROM THE SAME CASE SEEN FROM BEHIND. (Removal of the shoulder girdle.)

In all cases careful clinical observations are necessary, but they should not be extended over too long a period. If the skin over a rapidly growing tumor becomes red, the diagnosis must be made between a suppurative osteomyelitis and a gumma, for a tuberculous lesion, even after rupture into the soft tissues, does not increase rapidly in size, and a sarcoma, although containing a large number of vessels and sometimes causing fever, does not produce an inflammatory redness of the skin covering it. If, during the period of observation, potassium iodid has been administered without results, chronic suppurative osteomyelitis is the only diagnosis that can be made. If, on the other hand,

there is no inflammatory redness of the skin, a diagnosis of tumor may be made, and it may be regarded as a myelogenous form if a bony capsule can be demonstrated, either by palpation or the X-ray. Rapid growth speaks for a sarcoma and against benign tumors (fibroma, chondroma, echinococcus cysts of bone). It should, however, be clearly understood that many sarcomas have a relatively slow growth. If there is any suspicion that the lesion is of a sarcomatous nature, clinical observations should not be prolonged for more than one or two weeks. [In these cases an exploratory incision should be made early, and if the macroscopic appearances are characteristic enough to justify a diagnosis of sarcoma, the lesion should be removed. If the lesion is not sufficiently characteristic, tissue should be removed and examined microscopically, and then if the lesion is malignant, operative measures, unless there are contraindications, should be advised at once.]

FIG. 366.—SOFT, SPINDLE-CELL SARCOMA OF THE UPPER EPIPHYSIS OF THE HUMERUS REMOVED FROM A GIRL FOURTEEN YEARS OLD.

X-ray pictures frequently aid in making a diagnosis. Exostoses may be easily recognized, as they are conical or pedunculated, and their sharp outlines become continuous with those of the bone. In pictures of periosteal sarcomas, on the other hand, one sees irregular, cloudy shadows, the density of which depends upon the amount of bone the tumor contains. The boundaries of the tumor gradually become continuous with the outlines of the bone, which are indistinct at the points where the growth has reached the periosteum. In myelogenous sarcomas a faint shadow, sur-

rounded by a thin, expanded cortical layer of bone, may be seen when the bone surrounding the tumor has undergone pressure atrophy. The bright shadows merely indicate destruction of bone, and may be found in abscesses and cysts as well as in tumors (sarcoma, chondroma).

A delicate bony capsule, however, speaks against inflammatory foci of all kinds, even against tuberculosis, at least as it occurs in long, hollow bones, and the symmetrical shadows with indistinct boundaries against chondromas and cysts. Similar findings enable one to make a positive diagnosis in spontaneous fractures. If a central gumma is considered, the differential diagnosis can be easily made, as the gumma is always associated with irregular hyperostoses developing from the cortex.

The diagnosis is most difficult when a rapidly growing, soft, periosteal or myelogenous sarcoma invades an adjacent joint. As the tumor invades the joint capsule and para-articular tissues, a doughy swelling develops and the joint becomes fusiform in shape, suggesting a tuberculous or hæmophiliac lesion, the latter especially, as aspiration reveals blood. The X-ray findings are, as a rule, not definite enough to enable one to make a positive diagnosis. If a careful examination is made, a tumor attached to the bone near the joint may be palpated. Soon, however, the tumor extends beyond the joint, and the extremity enlarges as the result of circulatory disturbances. The enlargement is in marked contrast to the atrophy associated with tuberculosis of joints (Fig. 363).

In children the soft, myelogenous tumors, developing in the metaphysis, often cause a separation of the epiphysis. In these cases a diagnosis of suppurative osteomyelitis may be made, especially if the soft tumor masses fluctuate and there is fever.

It may be very difficult at times to differentiate between a tumor of this character and an aneurysm if the tumor develops adjacent to a large blood vessel (e. g., in the axillary fossa or popliteal space), for a telangiectatic sarcoma which pulsates may form.

Illustrative Cases to Demonstrate Difficulties in Diagnosis.—A few examples may be cited to illustrate how difficult it is at times to make a correct diagnosis.

A boy ten years of age fell ill with a swelling of the entire left arm. When first seen the arm was œdematous and the subcutaneous veins dilated. The cause of the venous stasis was a thickening of the left clavicle. This thickening, which gradually fused with the deeper structures, was covered by normal skin. The diagnosis rested between a chronic suppurative osteomyelitis and a sarcoma of the clavicle. After two weeks' observation the skin over the thickened area became slightly reddened and made the diagnosis of osteomyelitis positive. A focus of granulation tissue which communicated through a cloaca with the

interior of the bone was situated beneath the reddened skin. The pathology corresponded to that of the sclerotizing form of suppurative osteomyelitis. Permanent healing occurred after the newly formed bone was chiseled away and the granulation tissue removed.

A boy fifteen years of age fell ill with a painful swelling of the upper articular end of the left tibia. For two weeks the swelling had rapidly increased in size, especially anteriorly just below the tuberosity, where it was firmly attached to the skin. The temperature was about 98.8° F. X-ray pictures were negative. The diagnosis rested between a periosteal sarcoma and a superficial suppurating focus. The exploratory incision revealed a soft periosteal sarcoma.

Three weeks before examination a slightly painful, poorly defined swelling developed in the middle of the right leg over the fibula, in a man forty-five years of age. The skin was slightly adherent to the hard swelling, the contour of which became continuous with the fibula. Was the lesion a sarcoma, a suppurative osteomyelitis, or a gumma of the fibula? The X-ray pictures revealed an irregularly thickened fibula surrounded by a faint shadow cast by the mass. The thickening was not distinct enough to warrant a diagnosis of a gumma. After a week the skin over the swelling became reddened. A sarcoma could be excluded with certainty and a tentative diagnosis of a chronic suppurative osteomyelitis could be made. Just as an operation was about to be performed a round, white scar the size of a nickel, such as remains after the healing of a syphilitic ulcer, was found upon the other leg. The swelling disappeared completely under antisyphilitic treatment.

A woman thirty-five years of age suffered for a week with a swelling of the right wrist joint, which was supposed to be the result of a sprain. The joint was immobilized for two weeks, and when the dressings were removed the exudate had disappeared. Some slight thickening of the lower end of the radius which suggested some inflammatory lesion, such as tuberculosis or suppurative osteomyelitis, or a sarcoma could then be palpated. The X-ray pictures revealed a bright shadow surrounded by a thin layer of bone; therefore a suppurating focus could be excluded. The complete destruction of the spongy bone of the metaphysis and of the epiphysis spoke against tuberculosis.

The operation revealed a myelogenous giant-cell sarcoma about to rupture into the joint. The resected portion of the bone is reproduced in Fig. 362. The patient is well, and no recurrences have developed four years after the operation.

An emaciated man, twenty years of age, developed a painful swelling of the right knee soon after a fall. The joint was greatly swollen, and the normal outlines of the joint were lost as the capsule was filled with an exudate. The swelling was of a doughy consistency; fluctuation

and patellar balottement could be elicited only when considerable pressure was made. The entire clinical picture resembled closely that of tuberculosis. Aspiration revealed dark blood. It was possible, therefore, that the lesion might have been due to hæmophilia, and the patient gave a history suggestive of this disease. The joint was therefore immobilized for two weeks. After this time a soft swelling attached to the bone could be demonstrated, which extended upward above the condyle of the femur. X-ray pictures at this time were negative.

A diagnosis of sarcoma of the femur with rupture into the joint was made, which was verified by operation (amputation of the thigh). A soft myelogenous sarcoma which was very vascular and cystic had ruptured through the cortex at the point of attachment of the capsular ligament, and had first extended into the joint and later upward along the bone.

For other examples see chapter dealing with diagnosis of suppurative, tuberculous, and syphilitic lesions of bone.

Significance of Glandular Enlargement.—The glandular enlargements which occur in many cases of sarcoma of bone (Nasse) are not of much value in making an early diagnosis. The enlargements are frequently caused by the absorption of decomposition products from the tumor, and in the beginning they cannot be differentiated from inflammatory hyperplasias.

Prognosis.—The prognosis of sarcomas developing from bone is usually bad. Even the more benign forms gradually lead to a destruction of bone and the formation of metastases.

Indications for Treatment.—There is no question that sarcomas developing from bone or cartilage should be completely removed when there are no metastatic growths, unless they are so large or are so situated that removal is impossible. The extent of the operation that is necessary varies, as all of these tumors are not of the same degree of malignancy. The indication as to the extent of the operation that should be performed is dependent to a certain degree upon the character of the cells composing the tumor, as it is well known that giant-cell sarcomas represent the most benign, small, round-cell sarcomas the most malignant form of this class of tumors. The character and rapidity of the growth also determine, to some extent, the character of the operation. Well encapsulated tumors with an expansive growth—especially giant-cell sarcomas, which may be recognized by their brownish-red color—may be removed by blunt dissection with a periosteal elevator or shelled out with a sharp spoon. Experience has shown that even after such a conservative procedure as this the dangers of recurrence are not great. Usually, even in the treatment of encapsulated tumors, it is to be recommended that a part of the surrounding bone, the parent tissue

of the tumor, be removed. The part of the bone involved should be completely removed with a chisel, bone-cutting forceps, or saw, if the bony capsule is thin. If the tumor is situated upon one side of the bone, or is surrounded by a thick layer of the same, it may be completely removed and the continuity of the bone still be preserved by an osteal bridge.

Extensive resections of bone have given good results even in the treatment of central and periosteal sarcomas which have ruptured through their capsules. Of course the results following resection have only been good when the operation was performed before the tumor involved the soft tissues (von Bergmann, von Bramann, von Mikulicz, Nasse, and others). In the other forms of sarcoma nodules develop very early in the part of the bone adjacent to the primary focus after resection (König). The resected end of the bones may be approximated and held in apposition by silver wire or strong catgut sutures or a piece of dead bone may be placed between them. Of course the fragments should be immobilized until union is complete. If the tumor has already infiltrated the soft tissues, or if it is a soft, rapidly growing one, an amputation should be performed if it is situated upon an extremity; if the tumor occurs upon the trunk, neck, or head, it and the tissues adjacent should be removed—for example, if the sarcoma is in the orbit, the eye should be removed. In the treatment of large tumors of the thorax it is often necessary to open the abdominal and thoracic cavities.

Nasse has determined, in examining tissues from a number of osteosarcomas, that the cells frequently extend early in the clinical course of these growths along the blood vessels of the muscles. This imposes upon the surgeon not only the duty of operating as soon as possible, but also of removing all those muscles attached to the bone involved, even to their points of origin—that is, above the adjacent joint. Therefore this rule has been formulated, that in sarcomas of the forearm or leg a high amputation of the arm and thigh respectively should be performed; in sarcoma of the humerus the entire upper extremity including the shoulder girdle should be removed; in sarcoma of the femur a disarticulation at the hip should be performed with the removal of all the muscles passing from the pelvis to the femur.

[After operations for sarcoma the mixed toxins of prodigiosus and erysipelas (Coley) should be used as an insurance against recurrence.]

The *prognosis* of sarcomas developing from bone is bad. Reinhardt estimates that permanent recoveries occur in only 18 per cent of the cases. These unfavorable results are much more often due to metastases, especially in the lungs, than to local recurrences. The metastases often develop so rapidly after the operation that it is probable the cells

had already been deposited, but that the foci were not large enough to give rise to symptoms or demonstrable physical findings.

Concerning the treatment of inoperable sarcomas *vide* p. 775.

Chondro- and osteosarcomas developing from the soft tissues are of much less surgical importance than those arising from bone. They occasionally develop in fascia (Hammer), from the intermuscular connective tissue, in the mammary gland, the spermatic cord (Ribbert), and more frequently in mixed tumors.

LITERATURE.—*v. Franqué.* Ueber Sarcoma uteri. Zeitschr. f. Geburtsh. u. Gynäk., Bd. 40, 1899, p. 183.—*Friedrich.* Die Osteoplastik bei ausgedehnten operativen Diaphysendefekten der langen Röhrenknochen jugendlicher Individuen nach Entfernung bösartiger Knochengeschwülste. Zentralbl. f. Chir., 1904, No. 27, p. 26.—*Hammer.* Ueber ein malignes fasziales Riesenzellensarkom mit Knochenbildung. Beitr. z. klin. Chir., Bd. 31, 1901, p. 727.—*Jenckel.* Beitrag zur Kenntnis der Knochensarkome des Oberschenkels. Deutsche Zeitschr. f. Chir., Bd. 64, 1902, p. 66.—*Johannessen.* Sarcoma pelvis bei einem 11 Monate alten Mädchen. Jahresber. f. Kinderheilk., 1897.—*Kaposi.* Ueber einen Fall von sogen. Sarcomatosis cutis. Beitr. z. klin. Chir., Bd. 24, 1893, p. 526.—*W. Kramer.* Beitrag zur chir. Behandlung der bösartigen Sarkome der langen Röhrenknocken. Arch. f. klin. Chir., Bd. 66, 1902, p. 792.—*Kümmell.* Tumoren (Sarkome) der Wirbelsäule. Deutsche med. Wochenschr., 1902, Vereinsbeil, p. 131.—*Martens.* Zur Kenntnis der bösartigen Oberkiefergeschwülste. Deutsche Zeitschr. f. Chir., Bd. 44, 1897, p. 483.—*v. Mikulicz.* Ueber ausgedehnte Resektionen der lamen Röhrenknochen wegen maligner Geschwülste. Chir. Kongr.-Verhandl. 1895, II, p. 351. Disk. König, I, p. 104.—*R. F. Müller.* Zur Kenntnis der Fingergeschwülste. Arch. f. klin. Chir., Bd. 63, 1901, p. 348.—*Nasse.* Ueber einen Fall von multiplem, primärem Sarkom des Periostes. Virch. Arch., Bd. 94, 1883, p. 46;—Die Sarkome der langen Extremitätenknochen. Arch. f. klin. Chir., Bd. 39, 1889, p. 886;—Die Exstirpation der Schulter und ihre Bedeutung für die Behandlung der Sarkome des Humerus. v. Volkmanns Samml. klin. Vorträge, No. 86, 1893.—*Neufeld.* Kongenitales Osteosarkom des Schadels. Beitr. z. klin. Chir., Bd. 13, 1895, p. 730.—*Rehn.* Multiple Knochensarkome mit Ostitis deformans. Chir. Kongr.-Verhandl., 1904, II, p. 424.—*Reinhardt.* Sarkome der langen Röhrenknochen. Deutsche Zeitschr. f. Chir., Bd. 47, 1898, p. 525.—*Ribbert.* Beitr. z. Entstehung d. Geschwülste. Bonn, 1906.—*Schwimmer.* Remarks on Sarcoma of the Skin and its Varieties. Internat. Congress of Dermatology and Syphilography. Journal of Cutaneous and Genito-Urinary Diseases, 1897, April.—*Weisswange.* Primäre Sarkome der Wände der Schädelhöhle. I.-D. Tübingen, 1897.

(b) SARCOMAS COMPOSED OF CELLS RESEMBLING LYMPH CORPUSCLES

The term lymphosarcoma should be limited to those tumors which have their prototype in or develop from lymphatic tissue proper (lymph nodes, follicles in mucous membranes and the spleen). The term should no longer be applied to tumors developing from the connective tissue or reticulum of the lymphatic nodes, as these belong to the fibrosarcomas.

The distinction between lymphosarcoma and hyperplastic growths of the lymph nodes is not clear and sharp. Lymphosarcomas and the

specific enlargement of lymph nodes first described by Hodgkin in 1832, and called by a number of different terms, such as malignant lymphoma (Billroth), pseudoleukæmia (Cohnheim), adenie (Trousseau), aleukæmic malignant lymphoma (Orth), are often grouped under some generic term, such as malignant lymphoma (Orth) or lymphocytoma (Ribbert).

Lymphosarcomas.—Lymphosarcomas are composed principally of cells which resemble lymphocytes (therefore these tumors are called lymphocytomas by Ribbert). The cells are evenly distributed throughout a vascular reticulum covered by endothelium. The structure of the normal lymph node is not actually reproduced, as lymph follicles and lymph channels are wanting. The density of the reticulum varies, and so a distinction is made between the soft and hard forms.

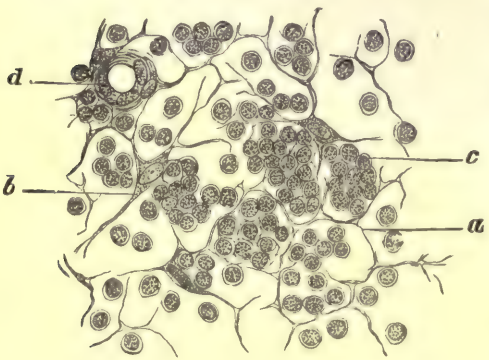


FIG. 367.—SECTION OF A LYMPHOSARCOMA OF THE NASAL MUCOUS MEMBRANE FROM WHICH MOST OF THE CELLS HAVE BEEN REMOVED BY SHAKING. *a*, Reticulum; *b*, cells of the reticulum; *c*, round cells; *d* (above and to the left), blood vessel lined with proliferating cells. (After Ziegler.)

These tumors always develop from normal lymphatic tissue, especially from the lymph nodes, the cervical, axillary, retroperitoneal, and mediastinal being most frequently involved. They also develop from the palatal and pharyngeal tonsils, the lymphatic follicles of the gastrointestinal tract, the thymus gland, the spleen, and bone marrow.

Age at Which These Tumors Develop and Characteristics.—Lymphosarcomas develop most frequently in young people from lymph nodes or other lymphatic structures to form rapidly growing tumors, sometimes soft, at other times hard, which rapidly break through the capsule of the lymph node and extend by way of the lymphatic channels to infiltrate the surrounding tissues. At times nodular tumors (which become fused with neighboring structures) develop. The skin covering the tumor may also become infiltrated. It has then at first a bluish color; later it becomes necrotic, and large ulcers, which may also become infected, develop.

Symptoms.—The symptoms produced by these tumors depend entirely upon their position. Mediastinal tumors, developing from the thymus gland or peribronchial lymph nodes, will compress the lungs and heart; tumors developing from the cervical lymph nodes may compress the trachea and œsophagus. These tumors frequently cause symptoms by pressing upon adjacent nerves and blood vessels.

If the tumor does not produce early death by pressure upon important organs, but continues to enlarge, metastases develop by way of the blood stream in the lungs, spleen, liver, and skin. These metastatic growths may appear as well-defined nodules or as diffuse infiltrations of an entire organ, sometimes being accompanied by alterations in the composition of the blood (lymphæmia). There are also cases in which the entire lymphatic system is involved, forming transitional stages between leukæmic lymphoma and aleukæmic malignant lymphoma (*vide* Coenen).

Diagnosis.—The diagnosis is based upon the rapid growth and position of the tumor. A lymphosarcoma cannot, however, be differentiated from other forms of sarcoma developing from lymph nodes or from a

FIG. 368.—LYMPHOSARCOMA OF THE NECK.

sarcoma of the soft tissues. In a lymphosarcoma the glands become fused, forming a single mass in which the separate lymph nodes can no longer be palpated, and the tumor breaks through the capsule invading the skin and surrounding structures, while in Hodgkin's disease

the glands remain isolated and there is no extension of the growth through the capsule to surrounding tissues.

Indications for Treatment.—Early and extensive removal of the growth is indicated, in spite of the fact that the results following even

Fig. 369.—Lymphosarcoma of the Neck and Left Axillary Fossa.

the most radical procedures are unsatisfactory, as recurrences rapidly develop.

Hodgkin's disease (malignant lymphoma) differs from lymphosarcoma, especially in its clinical course.

In this disease also there is a proliferation of lymphatic tissue, especially of the lymphatic nodes. The histological changes resemble very closely those found in lymphosarcoma, except that there is no tendency to disintegration.

Clinical Differences between Hodgkin's Disease and Lymphosarcoma.—As the disease progresses two distinct clinical differences between it

and lymphosarcoma may be noted: (1) In Hodgkin's disease the changes are limited to the lymph nodes, and the proliferating lymphatic tissue does not break through the capsules of the nodes and involve neighboring structures. (2) In Hodgkin's disease there is a progressive and successive involvement of different chains of lymph nodes, including the spleen, and in rare cases the bone marrow.

The development of foci in the liver, lungs, kidneys, and bone marrow would seem to indicate that malignant lymphoma is closely related

FIG. 370.—LYMPHOSARCOMA OF THE NECK. Rapid recurrence following operation. (From Professor Bevan's Surgical Clinic.)

to true tumors. It cannot, however, be demonstrated that these new foci are of metastatic origin, as it is possible that they may have developed from preëxisting lymphatic nodules.

Lymphatic leukæmia may resemble malignant lymphoma quite closely, as in this disease there is also a progressive enlargement of the lymph nodes with the formation of so-called heteroplastic nodules in different organs. A blood examination enables one to differentiate between the two very easily, as in lymphatic leukæmia the leucocytes are greatly increased, reaching 150,000 or more per cubic millimeter, and there is a great preponderance of lymphocytes which constitute from ninety to ninety-nine per cent of all the cells, while in malignant lymphoma the number of leucocytes is normal or only slightly increased.

Clinical Course and Appearance of Lymph Nodes.—Aleukæmic malignant lymphoma (Hodgkin's disease, pseudoleukæmia) develops most frequently in young and vigorous people. The disease begins with a gradual painless enlargement of the lymphatic nodes, the cervical group being most frequently, the axillary and inguinal less often, primarily affected. The separate nodes enlarge to form soft tumors, if there is connective-tissue induration to form hard ones, the size of a walnut or apple, and the entire chain of nodes becomes transformed into a nodular mass. The nodes are homogeneous and grayish-red in color upon section, the distinction between cortex and medulla being lost. There is no tendency to regressive changes or to break through the capsule of the lymph nodes and invade surrounding tissues; therefore the separate nodes can be palpated beneath the normal skin and displaced upon each other.

The growth is, as a rule, intermittent, periods of rapid growth alternating with periods during which the nodes remain stationary. When the proliferative changes extend to neighboring nodes, and the lymphatic tissues of the pharynx, gastrointestinal tract, spleen, and thymus gland become involved, general symptoms may develop. These consist of an *intermittent fever*, *increasing anæmia*, and *weakness*. Digestive disturbances (vomiting, diarrhea), secondary to involvement of the lymphatic tissue of the gastrointestinal tract, and interference with respiration and deglutition, secondary to proliferation of lymphatic tissue of the pharynx, may rapidly increase in severity and cause death, which in severe cases may occur in a few months.

Diagnosis.—The diagnosis is difficult so long as a single chain of lymph nodes is affected. An examination of the blood, with the absence of the characteristic changes of leukæmia, and the fact that the separate nodes may be palpated and moved freely beneath the skin and upon each other enable one to exclude lymphatic leukæmia and lymphosarcoma respectively. Lymph nodes the seat of gummatous lesions are hard and soon contract adhesions with the surrounding tissues and skin, leading to the formation of quite characteristic ulcers. The differential diagnosis between malignant lymphoma and tuberculous lymph nodes, espe-

cially when the latter are small and their characteristic changes but little pronounced, is often very difficult.

Tuberculous lymph nodes are more common in the young and are more frequently bilateral. Regressive changes are common in tuberculous nodes, leading to the formation of abscesses and fistulæ. The extent of the regressive changes varies, but, as a rule, the diagnosis can be made even before the nodes have contracted adhesions with surround-

FIG. 371.—MALIGNANT LYMPHOMA IN A WOMAN THIRTY YEARS OF AGE. (After Dietrich.)

ing tissues or have broken down to form tuberculous abscesses. There are, however, rare forms of tuberculosis of lymph nodes in which the entire lymphatic system is involved and in which softening does not occur. These hyperplastic forms of tuberculosis resemble very closely malignant lymphoma. In these cases a differential diagnosis based upon clinical data alone is impossible, and the tuberculous nature of the

lesions can be determined only by an accurate microscopical examination (demonstration of bacilli and giant-cells).

[Crowder has reported an exceedingly interesting case of generalized tuberculous lymphadenitis with the clinical and anatomical picture of Hodgkin's disease. In discussing this case he says: "The great number of terms applied to the condition designated as pseudoleukæmia, based upon the somewhat varied clinical course, as well as a limited variation in the gross and minute pathological changes and the greatly differing interpretation of these changes by different observers, are evidences of the heterogeneity of the class to which the name refers. The etiology is for the most part admittedly unknown; it is also admittedly various. The symptom complex determines the disease as the disease is now understood. Why, then, exclude those cases in which the tubercle bacillus is known to be the cause? A disease of known origin is not to be singled out and classed as a different disease, but as an etiological division of the heterogeneous class."]

It should not be considered, however, that there is any relation between hyperplastic tuberculosis of lymph nodes and Hodgkin's disease (Dietrich, Borst), even if cases have been reported in which the changes occurring in malignant lymphoma have been associated with tuberculosis of the lymph nodes and viscera (Ricker and others).

Treatment.—No attempt should be made at radical removal of the lymph nodes, even in the beginning of the disease, as there is a great tendency for the disease to recur and progress in other chains of lymph nodes. The internal administration of gradually increasing doses of arsenic in the form of Fowler's solution was recommended by Billroth, and in some cases it apparently has a favorable action. Decrease in the size of the lymph nodes and improvement of the general condition have been noted after daily injection of 3 or 4 minims of Fowler's solution into the enlarged lymph nodes (Czerny, von Winiwarter), after the injection of 3 or 4 minims of a one per cent solution of sodium arsenate into the subcutaneous tissues (von Ziemssen), and the use of the X-rays. Permanent recoveries after any line of treatment are apparently exceptionally rare. [A number of these cases have been much improved by the X-ray. The tumors grow smaller, and even disappear entirely. The treatment should not be pushed too rapidly, as with the breaking down and absorption of the tumor masses severe and even fatal toxæmia may occur.]

LITERATURE.—*Coenen.* Ueber ein Lymphosarkom der Thymus. Arch. f. klin. Chir., Bd. 73, 1904, p. 443.—*Dietrich.* Ueber die Beziehungen der malignen Lymphome zur Tuberkulose. Beitr. z. klin. Chir., Bd. 16, 1896, p. 377.—*Fr. Fischer.* Krankheiten der Lymphgefässe, Lymphdrüsen und Blutgefässe. Deutsche Chir., 1901.—*Ricker.* Ueber die Beziehungen zwischen Lymphosarkom und Tuberkulose. Arch. f. klin. Chir., Bd.

50, 1895, p. 573.—*Sternberg.* Universelle Primärerkrankungen des lymphatischen Apparates. Zentralbl. f. d. Grenzgeb., 1899, p. 641.—*Wilh. Türk.* Pseudoleukämie und Lymphosarkomatose. Wien. klin. Wochenschr., 1899, No. 40.—*v. Winiwarter.* Ueber das maligne Lymphom und das Lymphosarkom. Arch. f. klin. Chir., Bd. 18, 1875, p. 98.—*Yamasaki.* Zur Kenntnis der Hodgkinschen Krankheit und ihres Ueberganges in Sarkom. Zeitschr. f. Heilk., Bd. 25, 1904, p. 269.

Myelomas and Chloromas.—Myelomas and chloromas, which are rare and peculiar forms of tumors, should be classified with lymphosarcomas.

Multiple myelomas are rare. They appear as small, nodular, circumscribed, soft, grayish red growths, especially in the red marrow of different bones. They are regarded by some as lymphosarcomas, by others as localized hyperplasias of bone marrow. They have no tendency to infiltrate surrounding tissues or to form metastases, but remain limited to the bones primarily involved. The bone adjacent to a myeloma is, however, gradually destroyed and deformities of the spine, defects in the skull bones, and spontaneous fractures may occur.

The disease is most common in old people. Often it runs a rapid course with intermittent fever and symptoms of a severe primary anæmia, and terminates fatally.

It has frequently been demonstrated that a peculiar product known as Bence-Jones's albumose appears in the urine in multiple myelogenous osteosarcomas and myelomas. The reaction has been regarded as almost pathognomonic. Askanazy has, however, found the substance in the urine in a case of lymphatic leukæmia. He has come to the conclusion that Bence-Jones's albumose is indicative of some lesion of the bone marrow, most frequently of multiple myelomas, but also occasionally of other diffuse changes, such as occur in lymphatic leukæmia.

Literature.—*Hoffmann.* Ueber Myelomatose, Leukämie und Hodgkinische Krankh. Arch. f. klin. Chir., Bd. 79, 1906, p. 384.—*Wieland.* Studien über das primär multipel auftretende Lymphosarkom der Knochen. Virch. Arch., Bd. 166, 1901, p. 103.—*Winkler.* Das Myelom in anatomischer und klin. Beziehung. Virch. Arch., Bd. 161, 1900, p. 508.—*Yellinek.* Zur klin. Diagnose und path. Anatomie des multiplen Myeloms. Virch. Arch., Bd. 177, 1904, p. 96.—*v. Verelély.* Ueber das Myelom. Beitr. z. klin. Chir., Bd. 48, 1906, p. 614.

Chloromas are also rare. They appear as multiple new growths composed of lymphadenoid tissue. They are most common in children and young people, developing as firm, more rarely soft, tumors from the the periosteum of the bones of the skull and face (especially from the squamous and petrous portions of the temporal bone, the maxilla and orbits (both orbits usually being involved), also from the sternum, ribs, vertebræ, and long, hollow bones. They resemble quite closely very malignant sarcomas, in that they grow rapidly and form metastases in lymph nodes and the different viscera, but differ from sarcomas in that

the tumors have a bright green, yellowish, or grayish-green color, which is also reproduced in the metastatic growth. According to von Recklinghausen, the color is due to pigment formed by the cells; according to Chiari and Huber, to the fat contained in the tumors.

LITERATURE.—*Risel.* Zur Kenntnis des Chloroms. Deutsch. Arch. f. klin. Med., Bd. 72, 1902, p. 31.—*Rosenblath.* Ueber Chlorom und Leukämie. *Ibid.*, p. 1.

(c) SARCOMAS COMPOSED OF MYXOMATOUS TISSUE

Myxomas and Myxosarcomas.—These tumors are composed of soft, often indistinctly fluctuating, gelatinous masses of tissue. Their surfaces upon section appear yellow or grayish-red and transparent. A tenacious fluid containing mucin and permeated by delicate fibers may be removed from the cut surfaces of such a tumor.

The tissue composing these tumors closely resembles, histologically, embryonal connective tissue from which white fibrous tissue and fat

FIG. 372.—MYXOSARCOMA. *a*, Myxomatous tissue; *b*, columns of cells; *c*, fibrous tissue. (After Ziegler.)

are formed. It has its prototype in Wharton's jelly and the vitreous humor.

Histology.—The structure of these tumors is quite characteristic, polymorphous, stellate cells, provided with long processes, being found within a homogeneous, slightly granular or fibrillated ground substance, which is traversed by a relatively large number of blood vessels. Single giant-cells may be found in some parts of the tumor.

No tumor is ever composed of myxomatous tissue alone. It is usually

found in combination with other kinds of tumor tissue, especially with fat, cartilage, fibrous, and sarcomatous tissue. For this reason the tumors are called lipomyxomas, chondromyxomas, fibromyxomas, and myxosarcomas. If the tumor contains a large number of blood vessels it is called a myxoma teleangiectaticum or cavernosum; if the tissues undergo liquefaction and cyst formation the tumor is described as a myxoma cysticum.

Some connective-tissue tumors (fibroma of the nasal mucous membrane, pendulous fibroma or lipoma of the surface of the body) may become œdematous as the result of interference with their circulation and resemble myxomatous tissue, but these tumors should never be classified with myxomas. The delicate but distinct fibrillar ground substance, together with the microscopic mucin reaction (acetic acid), are the important characteristics of myxomas. Mucoid degeneration may occur in large tumors, such as chondromas, osteomas, fibromas, sarcomas, but in these cases there is, strictly speaking, no formation of myxomatous tissue. In order to signify this difference between the two forms, a fibroma, chondroma, or other tumor which has undergone secondary mucoid degeneration is called a fibroma- or chondroma-myxomatodes, etc., while a tumor which is composed of both myxomatous or fibrous tissue is called a myxofibroma or fibromyxoma.

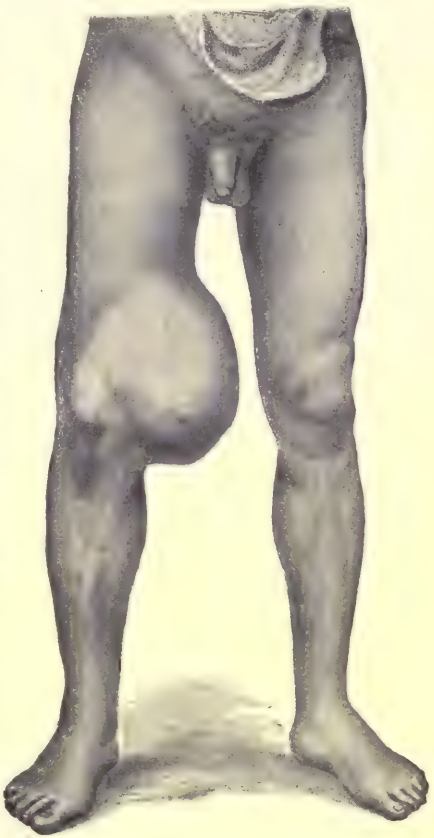


Fig. 373.—Myxosarcoma of the Fascia Lata.

Origin, Distribution, and Clinical Course.—These tumors are frequently of congenital origin. This indicates that they are the result of developmental disturbances occurring in embryonal life. Congenital tumors of this character have been observed in the cheeks by Zahn, in the remains of the umbilical cord by O. Weber, Kaufmann, and von Winckel, and in the mesentery by Borst. The displaced embryonal myxomatous tissues do not always form myxomatous tissue only, but fibrous and sarcomatous tissues as well.

Myxomas form nodular or lobulated tumors with well-developed connective-tissue septa. These tumors often attain considerable size. They are most common in young or middle-aged people, and their rate of growth varies. Some of these tumors have an expansive growth for a

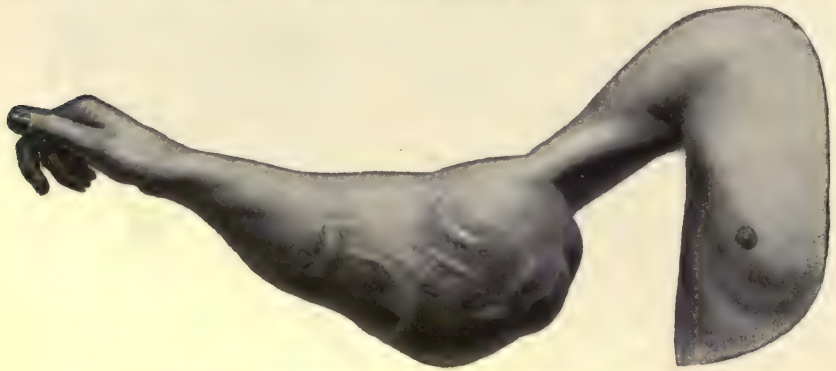


FIG. 374.—MYXOSARCOMA OF THE FASCIA OF THE ARM.

long time or permanently. The cellular tumor of a sarcomatous nature grows rapidly, breaks through its capsule, infiltrates the surrounding structure and skin, which ulcerates, and forms metastases. Some of these tumors are benign and some are malignant; depending upon their character, they are spoken of as myxomas or myxosarcomas.

These tumors may develop in a number of different tissues and organs, occurring most commonly in the cutaneous and subcutaneous, intermuscular and retroperitoneal connective tissues and fat, in bursæ and fascia, the periosteum and bone marrow, the membranes of the brain and spinal cord, the connective tissues of the nervous system and different organs (mammary gland, ovary, testicle, and spermatic cord, kidneys, liver, and lung).

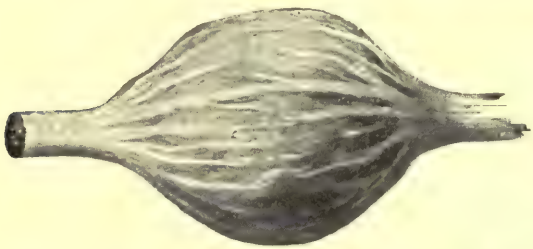


FIG. 375.—MYXOMA OF THE RADIAL NERVE IN A MAN THIRTY-FIVE YEARS OF AGE. Resection followed by nerve suture. Healing with complete return of function. No recurrence after five years.

They are most frequently found in the thigh, developing from the skin, the subcutaneous and intermuscular connective tissues, the fascia and the bursæ about the knee joint, less frequently upon the arm and in the gluteal region. They occur, when they develop upon the external genitalia, the neck, face, and scalp, as subcutaneous tumors. In the orbit they develop from the retrobulbar fat or the optic nerve. Myxo-

mas of the peripheral nerves, like fibromas, may occur as multiple tumors. When developing upon the nerves, they separate the nerve fibers and cause fusiform thickenings of the nerve trunks. In children these tumors may develop from the remains of the umbilical cord.

Myxomas of the bone marrow, which usually are cystic, may cause pressure necrosis and destruction of the compact bone surrounding them. Periosteal myxomas occur upon both the maxilla and mandible, usually in the form of encapsulated fibrous tumors. Tumors occurring within the jaw may develop from tooth buds, so long as they consist of myxomatous tissue, and appear as a variety of odontoma. Myxomas of the endocardium have a special significance. They appear as lobulated tumors, developing into the left auricle from the interauricular septum. More rarely they are situated upon the valves. Small pieces of these tumors may become separated, causing embolism (*vide* Jacobsthal). Myxomatous tissue is also frequently found in mixed tumors, especially in those occurring in the parotid gland.

Fig. 376.—Retroperitoneal Myxosarcoma and Intermuscular Myxosarcoma of the Gluteal Region (not Connected with Nerves). Multiple soft fibroma of the skin and many pigmented moles.

Naturally, the clinical symptoms caused by these tumors depend upon their position, the character of their growth, and the size they attain.

Diagnosis.—These tumors have a characteristic consistency, and the diagnosis is not difficult if they have grown rapidly and infiltrated the tissues. The consistency, combined with the character of the growth, generally enables one to make a diagnosis of a myxosarcoma. The diagnosis is more difficult if the tumor is encapsulated, as then a myxoma may resemble a tuberculous abscess, a lipoma, or any variety of cyst. Sometimes aspiration reveals a small amount of fluid containing delicate fibrillæ, and in that event ranula, hygromas, and cysts, which may contain mucoid, gelatinous masses, must be excluded. As a rule this can easily be done, when the position of the tumor is considered.

Treatment.—The treatment to be instituted depends upon whether the tumor is encapsulated or not, and upon whether it has grown slowly or rapidly. If the tumor is encapsulated and has grown slowly, enucleation is all that is required. If, on the other hand, the tumor has grown rapidly and infiltrated surrounding structures, a radical operation must be performed. In tumors of the extremity an amputation may be necessary. If a myxoma develops upon a nerve, the latter should be resected and nerve suture performed, if the tumor cannot be enucleated. In myxomas of the optic nerve, the bulb of the eye may be retained, unless the tumor has already extended to it.

LITERATURE.—*Jacobsthal.* Primäres Fibromyxom des linken Vorhofs. Virch. Arch., Bd. 159, 1900, p. 351.—*Orth.* Schleim und Schleimgeschwülste. Gesellsch. d. Wissensch. zu Göttingen, 1895.—*Rumler.* Ueber Myxom und Schleimgewebe. I.-D., Bonn, 1881.—*v. Winckel.* Ueber angeborene solide Geschwülste des perennierenden Teiles der Nabelschnur. v. Volkmanns Samml. klin. Vortr., No. 140, 1895.—*Zahn.* Ueber ein Myxosarkom bei einem 6monatl. Kinde, hervorgegangen aus dem Saugpolster der linken Wange. Deutsche Zeitschr. f. Chir., Bd. 22, 1885, p. 387.

(d) SARCOMAS COMPOSED OF PIGMENT CELLS

Melanosarcomas.—Sarcomas composed of pigment cells are called melanomas (malignant melanomas, melanosarcomas, chromatophoromas).

Most Important Characteristics.—The most important characteristics of these tumors, which occur primarily only in the eye and skin and the adjacent mucous membranes, are their color and malignancy. Developing at any age, they form round, nodular, pedunculated or fungiform growths which in the beginning are covered by a delicate epidermis but soon ulcerate. They are very vascular, bleeding easily, and are covered with crusts or become transformed into craterlike ulcers. The floor of the ulcer, which is black or brownish black in color, appears as if filled with india ink and secretes a serohæmorrhagic, usually black, fluid.

The consistency of these tumors varies; sometimes they are hard, at other times soft. The color of the tissues—which varies between a black and yellowish-brown shade, is frequently apparent upon the surface, and can always be seen after section—indicates the character of the tumor. The pigment is either distributed evenly throughout the tumor tissues or occurs in areas which may be surrounded by tissues absolutely devoid of pigment. Hæmorrhagic foci, which are frequent in tumors of this character, may be distinguished from the pigmented tumor tissue by their dark, brownish-red color.

Regressive changes in the tumor tissue, ending in liquefaction, lead to the formation of large cavities filled with black fluid.

Melanomas of the eye develop from the choroid, the iris, or pig-

mented areas along the margin of the conjunctiva. Melanomas developing in the eye grow rapidly after rupture of the sclera or cornea, destroying the bulb, and form large, nodular, pigmented growths, which protrude from the orbit or extend into the cranial cavity.

Most of the melanomas developing in the skin begin in congenital pigmented areas or in wartlike moles. When they develop, there appears at the site of the mole a brownish or brownish black nodule, which in the beginning develops slowly, sometimes, however, after some irritation, such as cauterization, very rapidly. The surface of the nodule ulcerates, becomes fissured, and bleeds easily. The primary tumor may scarcely have reached the size of a walnut when small, secondary nodules, which appear as small bluish points, develop in the skin surrounding the tumor. These, as they enlarge, fuse with the primary growth, contributing in this way to its rapid increase in size.

FIG. 377. — MELANOSARCOMA OF THE FOOT IN A MAN FIFTY YEARS OF AGE. Small secondary nodules may be seen in the skin surrounding the primary tumor.

Early Involvement of Lymph Nodes.—The regional lymph nodes enlarge early, and upon section black or brown areas, corresponding to metastatic growths, may be seen. Alteration of the functions of the viscera, general weakness, and an increasing anæmia indicate the formation of metastatic growths by way of the blood stream. Extensive metastases develop exceedingly rapidly and make melanomas the most malignant of all tumors.

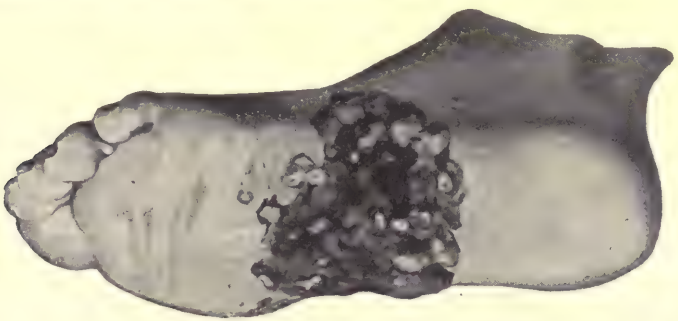


FIG. 378.—MELANOSARCOMA OF THE FOOT IN A WOMAN FORTY-FIVE YEARS OF AGE.

Most Common Sites for Development.—As pigmented moles may occur upon any part of the skin, so melanomas may develop any-

where upon the surface of the body. They develop most frequently, however, in the skin of the face and of the extremities. In the extremities, the flexor surfaces, the bed of the nail and the tissues adjacent to it are most commonly involved (Fig. 379). In rare cases these tumors develop in the mucous membrane of the rectum, nose, and soft palate. It is doubtful whether the arachnoid and pia mater are ever primarily involved, for secondary nodules may develop in those membranes many years after an eye, the seat of such a tumor, has been enucleated (Dobbertin), and unless an accurate history can be obtained, secondary nodules may be regarded as a primary tumor.

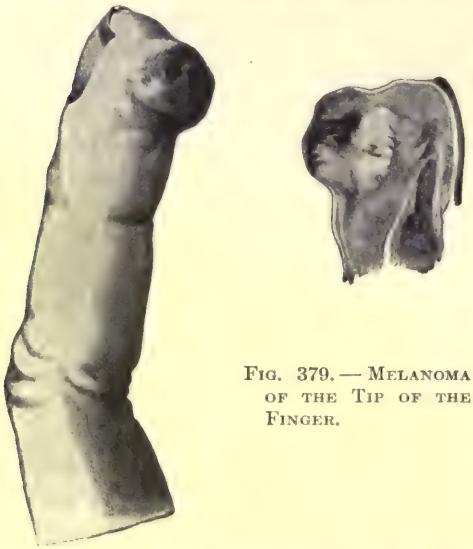


FIG. 379. — MELANOMA OF THE TIP OF THE FINGER.

Character of the Pigment.—The pigment *melanin* occurs as a granular or lumpy brown mass within the cytoplasm, and is to be regarded as a product of secretion of the cells. Usually the pigment lies around the periphery of the cell body. If regressive changes occur, the pigment is set free and is then taken up by wandering cells to be transferred to other parts of the tumor (Borst).

Origin of Melanosarcomas and Nature of Cells.—It is probable that melanomas develop from displaced groups of pigment cells. The occurrence of these tumors in the sclera and retrobulbar fat may be satisfactorily explained in this way. Another fact which supports this theory is that melanomas of the skin develop in congenital pigmented nævi. Therefore there must be some relation between the cells composing melanomas and those found in pigmented nævi. Nævi contain pigment cells in the lower layers of the epidermis; well-developed chromatophores in the corium, which is thickened in the warty forms; and groups and columns of round or oval nonpigmented cells, the significance of which has been differently interpreted. These cells have been regarded as epithelium, as endothelium of the lymphatic spaces, and as connective-tissue cells, which were related to the cutaneous nerves. Ribbert believes that they are genetically chromatophores which have not gone on to complete differentiation (without pigment).

According to Virchow, melanotic tumors are composed of groups of

cells which carry pigment, and only after regressive changes is the pigment found without the cells. According to Ribbert, the cells found in melanomas belong to a definite type, the pigmented connective-tissue cell or chromatophore; therefore, the term chromatophoroma.

The pigment cells found in a melanoma of the bulb differ in early stages of the growth very little from the normal chromatophores, as they occur in the choroid, the iris, and in pigmented areas in the skin, such as the nipple, the skin about the anus, and in congenital pigmented moles. Elongated cells with long processes lie close together, arranged in bundles, and the tissue resembles histologically a spindle-cell sarcoma. If a rapidly growing tumor of the choroid breaks through the sclera, it assumes, according to Ribbert, the structure common to melanomas of the skin. In these cases normal chromatophores can be demonstrated only in fresh preparations. Upon microscopic examination of these rapidly growing tumors of the choroid and of melanomas of the skin, large round, spindle, and polymorphous cells with short processes are

FIG. 380.—METASTASES (FROM A MELANOSARCOMA) IN THE SMALL INTESTINE AND MESENTERY.

found. Some of these contain pigment, while others are pigment free. According to Ribbert, these differences in the cells are dependent upon different degrees of development. He regards the cells without pigment as young forms, the large round, heavily pigmented cells as degenerating forms, and the other cells as more or less well-developed cells, which resemble epithelium and lie within a fibrillar network. Upon microscopic examination an alveolar arrangement is seen. The blood vessels lie within this fibrillar network. The cells which contain the most pigment are adjacent to them. The last fact is the more striking as the cells lying in the pseudo-alveoli usually are pigment free, being pigmented in the very dark tumors only. Fine fibrillæ from the bundles of fibers pass between the different cells. It is important to

determine whether the fibrillæ are processes of the pigmented cells, for if they are, it speaks against the origin of melanomas from pigmented epithelial cells of the skin and retina. [Notwithstanding the number of investigations that have been made regarding the origin of chromatophores, there is but little uniformity of opinion at the present time as to whether they are derived from mesoblast or epiblast. Many pathologists now follow Unna and regard the cells found in melanomas as derivatives of down growths of surface epithelium and classify the tumors as carcinomas. These tumors should probably be called melanocarcinomas instead of melanosarcomas.] Clinically, these tumors differ in some ways from sarcomas, probably the most striking difference being the frequency with which they, like carcinomas, form metastases by way of the lymph stream.

It is not clear in what way melanomas develop from pigmented nævi. According to Ribbert, they develop only from chromatophores. Others believe that they develop from groups of nævus cells. Krompecher believes that they develop from the basal cells of the epidermis, which, he thinks, may also proliferate to form nævus cells.

It is a striking fact in animal pathology that melanomas are very common in white horses. This can only be interpreted as indicating that irregularities in the distribution and arrangement of pigment cells, resulting in tumor formation, are more common in those animals in which the pigmentary processes and changes are most marked.

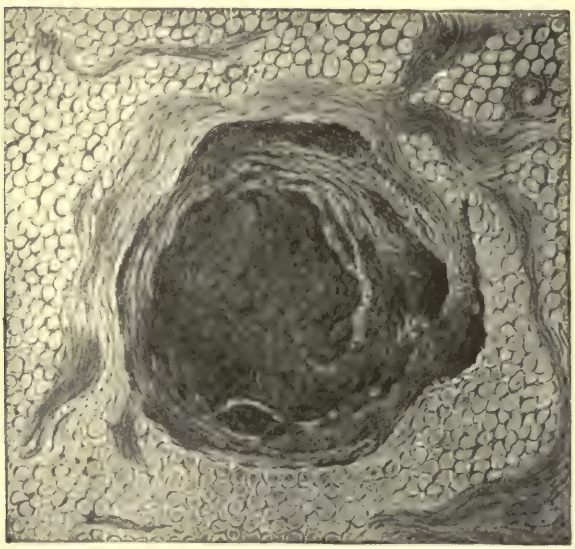


Fig. 381.—Melanotic Nodule in the Subcutaneous Fat (20 cm. from the primary tumor).

Mode of Growth; Metastases. — Melanomas, which usually are composed of poorly differentiated elements, always have an infiltrating growth, the cells invading the tissue spaces. The lymphatic vessels and blood vessels are frequently involved, and the lumina of the vessels adjacent to the tumor may be filled for some distance with proliferating cells.

The cells invade the lymphatics surrounding the tumor, leading to the formation of small secondary nodules. Not infrequently secondary nodules are encountered in the subcutaneous tissues during the excision of a melanoma. If these are found, the excision must be carried well beyond the apparent boundaries of the tumor into healthy tissues. The tumor cells are frequently carried by the lymphatic vessels to the regional lymphatic nodes, which become enlarged, forming large nodular tumors. In melanoma of the bulb of the eye, the first metastases frequently develop in the brain.

The invasion of the blood vessels is followed by embolism and the development of extensive metastases. In some cases so many emboli are discharged that pigmented tumor cells may be easily demonstrated in the capillaries and in the larger vessels of the viscera, especially in the liver. The metastases developing in a melanoma may be very extensive, appearing as closely grouped nodules, not only in one, but in many or all of the organs (lungs, liver, spleen, kidneys, brain, heart, intestinal wall, serous membranes, bone marrow, skin). Besides there may be an extensive discoloration of the skin and mucous membranes (melanosis).

The amount of pigment contained in the metastatic growths varies. Adjacent to dark, black foci, may be areas and nodules which contain little or no pigment. A rapid proliferation of the cells is associated with a decrease in the amount of pigment formed.

The pigment set free when regressive changes occur in a melanoma may gain access to the blood stream. Part of the pigment is deposited in the different organs, and part is dissolved to be excreted in the urine (melanuria), which then assumes a dark color.

Clinical Course.—The clinical course of melanomas depends upon their rapidity of growth and upon the number of metastases which form. Non-operated cases die in a short time of anæmia, metastases and general infections developing from ulcerating and infected tumors. Recurrences are very frequent after operation. The early development of metastases after an operation indicates that emboli had lodged in the different viscera before the operation was performed. A fatal termination may at least be delayed by the removal of the tumor, and sometimes the first metastases develop years afterwards (Dobbertin). A permanent cure can be expected only when the operation is performed very early.

Diagnosis.—The diagnosis of melanomas in the early stages may offer some difficulties. A melanoma should never be mistaken for a benign tumor, as the former grows rapidly. A vascular sarcoma with extensive hæmorrhages may resemble a melanoma macroscopically, but a microscopic examination enables one to differentiate between the two.

Indication for Treatment.—The indication for treatment, taking into consideration the malignancy of these tumors, is to remove the growth as early and thoroughly as possible. The regional lymphatic nodes should be removed, even if not enlarged. [It should be remembered that melanomas form early and extensive lymphatic metastases, differing in this respect from other forms of sarcomas, and that if the operation is to be at all successful the lymphatic nodes draining the primary tumor must be radically removed.]

In a melanoma of the bulb of the eye, the contents of the orbit should be removed, even if the growth is still confined to the eye. In a melanoma of the skin, excision should be carried wide of the tumor into healthy tissues and down to muscle. If the tumor occurs upon the extremity, amputation at some distance from the growth should be performed, unless there are contraindications.

Nævi.—The classification of nævi is still a mooted question. Von Recklinghausen and Ziegler regard them as pigmented forms of lymph-

Fig. 382.—Nævus Pigmentosus Pilosus.

angiomas. Unna and others regard them as epithelial tumors. Borst classifies them as melanotic fibromas. Following Ribbert's example, we will classify them under melanomas. There are several reasons justifying this classification. In the first place, melanomas frequently develop

from pigmented nævi, and the fact that both may be pigmented indicates that there is a certain relationship between the two.

Pigmented nævi may appear in a number of different forms, between which there may be a number of transitions.

Nævi Spili.—The flat nævi (nævi spili) appear as sharply defined, irregular, coffee-brown or black areas in the skin, varying in size from

Fig. 383.—Nævus Pigmentosus Verrucosus.

the head of a pin to a saucer. They are covered by a smooth epidermis and do not extend above the level of the skin. They are very similar in gross appearance and in structure to lentigines and freckles, the latter developing upon the face and the dorsum of the hands after exposure to the sun's rays. The flat, smooth nævi may be associated with elephantiasis of the nerves, and may then be distributed with considerable regularity over the entire surface of the body (*vide* p. 794).

Nævi Prominentes.—The elevated nævi (nævi prominentes) are likewise sharply defined. They occur as deeply pigmented, beetlike, soft growths, which often become very large. In some cases they may extend over the entire trunk. Their surfaces may be either smooth and shining, or covered with shallow furrows, corresponding to folds in the skin, or with a thick hair and warty growths. The following forms are differentiated, depending upon the character of the surface of the nævus: (*a*) Hairy moles (nævi pilosi, Fig. 382) are covered by a thick

growth of short dark hair, which is sometimes soft, at other times stiff. The hair, together with the brown discoloration of the mole, is suggestive of an animal's hide. (*b*) Warty nævi (nævi verrucosi, papillomatosi) are covered by warty growths. They appear either as small, soft, or if cornification is marked, as hard, round or pedunculated nodules, or as long, thorny papillæ (Fig. 383). The papillomatous growths often develop later upon a preëxisting nævus.

Fig. 384.—Congenital Hairy Nævus. In the upper parts of the nævus papillary growths, which have been developing for some years, may be seen.

Origin of Pigmented Nævi.—Pigmented nævi are of congenital origin, or appear shortly after birth. They enlarge slightly and slowly until the full growth of the individual is attained, and then they remain of the same size and form. Warty growths may, however, develop upon a nævus in later life (Fig. 384).

They may occur upon any part of the surface of the body, frequently being multiple (Fig. 385). They cause no symptoms.

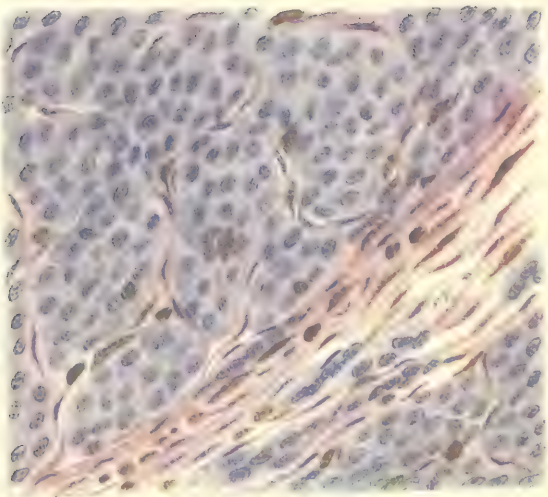
In many cases nævi occur along the course of cutaneous nerves, having a unilateral or symmetrical distribution (nerve nævi, neuropathic papilloma, occurring especially in the face and neck), or in the folds and growths of skin covering plexiform neuromas and superficial neurofibromas. Flat nævi are also frequently associated with soft warts and fibromas of the skin, being distributed over the entire surface of the body. All these facts seem to indicate that nævi have some relation to nerves. Their relation to cutaneous nerves and neurofibromas is not clear. Soldan regards nævi as fibromas of the most delicate cutaneous nerves, but his findings have not as yet been verified.

Histology of Nævi.—Histologically, the elevated nævi are composed of the proliferated connective tissue of the cutis, especially of the papillæ, which are no longer well defined against the subcutaneous tissues. The epidermis is also involved in the growth, so that pigmented nævi are closely related to fibroepithelial tumors.

1. Flat Pigmented Nævus.

(*a*) Groups of nævus cells and chromatophores in the connective tissue.

(*b*) Groups of pigment cells within and beneath the epidermis.



2. Alveolar Melanoma of the Skin.

3. Mixed Tumor of the Parotid Gland.

(*a*) Cartilage.

(*b*) Stroma resembling fibrous tissue.

(*d*) Columns of epithelial cells with glandlike structure and hyaline contents.

(*e*) Interstitial substance resembling a sarcoma.

The pigment lies in fine, granular, brownish masses in the cylindrical cells of the stratum germinativum and in the large chromatophores distributed throughout the corium. Within the proliferated connective tissues of the cutis are found groups and columns of round or oval cells, the so-called *nævus cell clusters.* These cells, together with the pigment cells, are the most important constituents of the flat nævi. According to von Recklinghausen, Ziegler, and Borst, the so-called nævus cells are the proliferated endothelium of the lymphatic vessels; according to Ribbert, they are imperfectly differentiated chromatophores surrounded by a fine fibrillar network. Soldan regards them as connective-tissue cells. Marchand, Orth, Unna and his school believe that these cells are of epithelial origin, while Krompecher believes that they are derived from the basal cells of the epidermis. These groups of cells often occur in columns, or are radially arranged, passing toward the surface of the nævus. In the deeper parts the groups of nævus cells are closer together than they are near the epidermis, upon the cells of which they may rest.

Fig. 385.—A Young Woman Eighteen Years of Age with Multiple Nævi and an Elevated, Hairy Verrucous Nævus which Involves the Left Cheek, Scalp, and Neck.

Diagnosis. — The diagnosis of pigmented nævi may be made without any difficulty. The projecting forms often suggest that a plexiform neuroma may be present in the deeper tissues (*vide* p. 794).

Treatment. — Nævi are removed especially for cosmetic purposes, and then, as a rule, only when they occur upon the face and neck. In removing a nævus the skin surrounding it should be circumscribed and then dissected away from the subcutaneous tissues. If the defect is

so large that the wound cannot be closed by sutures, it should be covered by skin grafts. Pedunculated flaps should not be used unless it is absolutely necessary, for they leave disfiguring scars and large secondary defects. Epidermal strips should not be used in skin-grafting these defects, as disfiguring tumorlike masses of scar tissue may form which are less desirable than the nævus. If a single, large, non-pedunculated cutis flap is used a beautiful result may be obtained. Particular care should be exercised in excising a nævus of the eyelid and in grafting the defect. If the nævi are very widely distributed, excision should be limited to those upon the face or upon areas uncovered by clothing.

The development of nodular growths upon a nævus is the second indication for operation. Every nævus is potentially a malignant tumor —a melanoma. If the development of nodules or rapid growth arouses suspicion of malignancy, thorough removal should not be delayed.

Xanthomas.—The growths occurring in the skin known as xanthomas or xanthelasmas consist of small, sulphur-yellow or brown, circumscribed areas (xanthoma planum), and nodular elevations (xanthoma tuberosum). They occur most frequently in the skin, especially upon the eyelids, but also in other parts. They are also found upon the mucous membranes of the respiratory passages, mouth, and œsophagus. These growths are often multiple, occasionally congenital. The acquired forms are most common in old age.

These growths contain cell nests which resemble those found in nævi. They differ from these, however, in that they contain a granular, yellow pigment and fat droplets. The cells containing fat droplets resemble closely those found in proliferating fatty tissue (Borst).

LITERATURE.—*Aschenbach.* Ein Fall von orbitalem Melanosarkom. Virchows Arch., Bd. 143, 1896, p. 324.—*Borst.* Die Lehre von den Geschwülsten. Wiesbaden, 1902, pp. 943 and 960.—*Dobbertin.* Beitrag zur Kasuistik der Geschwülste. Zieglers Beiträge zur path. Anat., Bd. 28, 1900, p. 42;—Melanosarkom des Kleinhirnes und des Rückenmarks. *Ibid.*, p. 52.—*Just.* Ueber die Verbreitung der melanot. Geschwülste im Lymphgefässystem. I.-D., Strassburg, 1888.—*Krompecher.* Der Basalzellenkrebs. Jena, 1903, p. 100 (Nævi).—*Joh. Kröner.* Ein ausgedehnter Fall von Papilloma neuropathicum. I.-D., Würzburg, 1890.—*Lanz.* Experim. Beitrag zur Frage der Uebertragbarkeit melanot. Geschwülste. Kochers Festschrift. Wiesbaden, 1891.—*Martens.* Ein Beitrag zur Entwicklung des Melanosarkoms der Chorioidea bei angeborener Melanosis sclerae. Virchows Arch., Bd. 138, 1894, p. 111.—*Putiata-Kerschbaumer.* Das Sarkom des Auges. Wiesbaden, 1900.—*Ribbert.* Das Melanosarkom. Zieglers Beitr. zur path. Anat., Bd. 21, 1897, p. 471;—Geschwulstlehre. Bonn, 1904.—*Soldan.* Ueber die Beziehungen der Pigmentmäler zur Neurofibromatose. Arch. f. klin. Chir., Bd. 59, 1899, p. 261.—*Unna.* Die Histopathologie der Haut., 1894;—Naevi und Naevokarzinome. Berl. klin. Wochenschr., 1893.—*Wagner.* 19 Fälle von Melanosarkom. Münch. med. Wochenschr., 1887, p. 14.—*Wiener.* Ueber ein Melanosarkom des Rektums und die melanot. Geschwülste im allgemeinen. Zieglers Beitr. zur path. Anat., Bd. 25, 1899, p. 322.—*Williams.* Beiträge zur Histologie und Histogenese des Uterussarkoms. Zeitschr. f. Heilk., Bd. 15, 1894, p. 141.

B. TUMORS COMPOSED OF MUSCLE

Tumors composed mostly of muscle fibers are called myomas. A *leiomyoma* composed of smooth muscle fibers is differentiated from a *rhabdomyoma* composed of striated muscle fibers.

CHAPTER I

LEIOMYOMAS

THE leiomyoma is an especially benign tumor. It is encapsulated and rarely has an infiltrating growth or forms metastases. They occur as round tumors with a smooth or nodular surface, most frequently in the uterus, less often in the muscular layers of the alimentary canal and urinary tract. The tumors occurring in the uterus (which are frequently multiple) and in the intestine grow slowly and may become as large as a man's head. Upon section, they have a grayish-red color, and the irregular, thick network of wavy tissue bundles is even more pronounced than in fibromas.

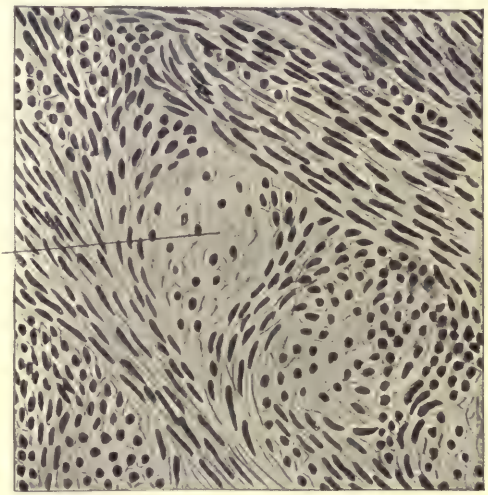


FIG. 383.—LEIOMYOMA OF THE UTERUS. *a*, Transverse section of bundle of muscle fibers.

Histology. — Upon histological examination, longitudinal, oblique, and transverse bundles of smooth muscle fibers with rodlike nuclei may be seen. The muscle fibers have blunt, rounded ends and may be easily differentiated from the pointed connective-tissue fibers found in fibromas. Between the bundles of muscle fibers lie varying amounts of fibrous tissue which carries the blood vessels. The fibrous tissue may encircle the bundles of smooth muscle fibers or run parallel with them. The tumor is either surrounded by a thin capsule, being sharply delimited from the surrounding tissues, or bundles of muscle fibers extend into the surrounding tissues, and then the tumor is firmly attached.

Consistency.—If there is but little fibrous tissue, the tumor is soft and succulent; if, however, it is well developed, the tumor has

the consistency of a fibroma (fibromyoma). A large number of dilated vessels may also be present, and the tumor is then called a *fibromyoma teleangiectaticum*. Malignant myomas are very cellular. They resemble spindle-cell sarcomas, as they grow rapidly, infiltrate surrounding tissues, and may form metastases. It cannot be positively determined in these malignant myomas whether the sarcoma develops from the fibrous tissues of the tumor or from the muscle fibers proper.

Regressive Changes.—In the large tumors all the regressive changes may be found which follow an insufficient blood supply. Large cavities filled with detritus, areas that have undergone hyaline degeneration or have become calcified, and in pedunculated myomas an œdematous infiltration, the result of venous stasis following torsion or kinking of the pedicle, may be seen.

Fig. 387.—Uterus with Subserous Myoma Removed from a Patient Thirty-nine Years of Age, Frontal Section.

Origin of Leiomyomas.—Nothing but conjectures, based upon the occasional finding of glandular elements in myomas of the uterus and intestinal wall, can be made concerning the genesis of leiomyomas. Apparently all myomas, certainly the adenomyomas containing epithelial tubules, are the result of developmental disturbances. Pieces of muscle become separated from their normal connections early in embryonal life, which assume an independent growth and form tumors later (Ribbert).

Most Common Sites for Development.—Leiomyomas are most common in the uterus. A large number may develop in the fundus, being present in early life as small growths. They may be situated beneath the mucous or serous membrane (submucous or subserous fibromyomas) or in the uterine wall (intramural fibromyomas). The submucous and

subserous forms are frequently pedunculated. They form round or hemispherical encapsulated tumors of firmer consistency than the uterine musculature, and cause a number of different symptoms (severe menorrhagia or metrorrhagia being the most prominent). Fibromyomas of

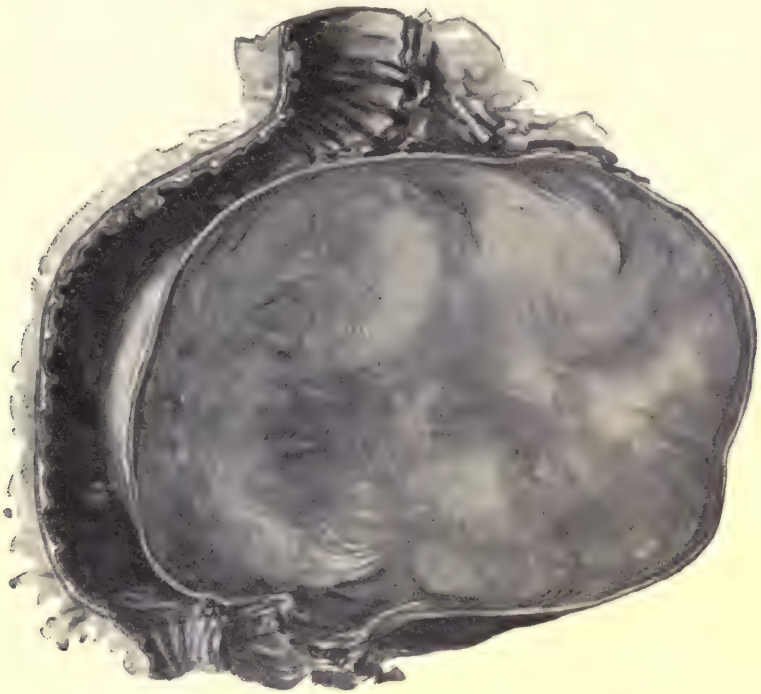


Fig. 388.—Fibromyoma of the Posterior Wall of the Rectum Removed by Resection of the Rectum (Male Patient Thirty-five Years of Age). (Lexer.)

the uterus interfere with pregnancy and labor, and may also become infected and undergo putrefactive changes. The ligaments and tubes are most frequently involved after the uterus.

Adenomyomas.—It is important in discussing the genesis of myomas of the uterus to note that von Recklinghausen has demonstrated tubules and cavities lined with cubical, cylindrical, and ciliated epithelium within the tumor tissue in some cases. These adenomyomas are usually small, poorly defined, subserous tumors, occurring most frequently upon the posterior surface of the uterus at the junction of the tubes with the uterus, in the broad and round ligaments. It is supposed that some of these tumors develop from displaced mucous glands, some from the Wolffian body or duct, and some from Müller's duct, pieces of muscle being displaced with the epithelium.

Leiomyomas of the Intestines, Œsophagus, Urinary Passages, etc.—Leiomyomas are found less frequently in the gastrointestinal tract, developing from the musculature.

Myomas developing in different parts of the œsophagus usually remain small and cause no symptoms. Myomas of the stomach, of the small and large intestine and rectum grow slowly. They may become as large as a man's head. These tumors may be sessile or pedunculated. Sometimes they grow into the lumen of the bowel, while at other times they develop upon the outer side. They may give rise to a number of

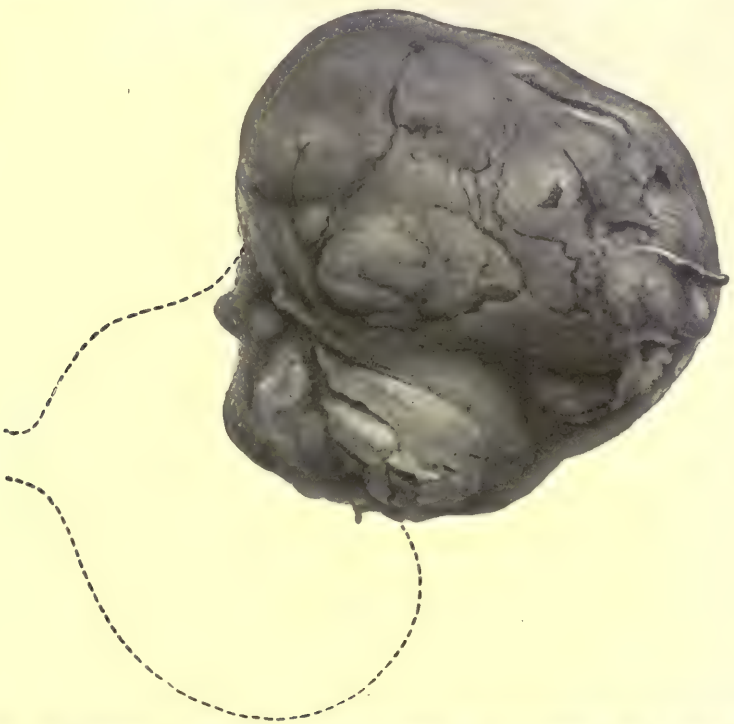


FIG. 389.—MALIGNANT LEIOMYOMA OF THE BLADDER. (Male patient, sixty years of age.) The subserous myoma which was attached to the bladder wall by a broad base had developed into the abdominal cavity. It was necessary to resect the wall of the bladder during the removal of the tumor. No recurrence after four years. (Lexer.) Dotted line indicates the outline of the bladder.

different symptoms (intestinal obstruction, intussusception, hæmorrhage after ulceration of the mucous membrane). The finding of pancreas tissue in a myoma of the stomach (Cohen) indicates that these tumors, like adenomyomas of the uterus, develop from a congenital anlage. Malignant myomas are very rare.

Myomas of the urinary tract are not very common. Up to the pres-

ent time only seventeen cases have been described. They develop from the musculature and may grow into the cavity of the bladder or externally (Fig. 389), attaining considerable size. Büttner found a large myoma in the urethra. Small myomas which were attached to the fibrous capsule have been found in the kidneys.

Pure myomas are rarely found in the prostate. Usually a part or all of the gland becomes hypertrophied, all of the elements being involved.

Myomas occasionally occur in the mammary glands and testicle. Myomas of the skin occur upon the trunk and extremities. They appear as small multiple nodules of firm consistency which project above the surface. These tumors, covered by normal skin, never become larger than a hazelnut. They frequently give rise to severe pain. They develop from the musculature of the cutaneous blood vessels or hair follicles. Clinically they cannot be differentiated from fibromas of the cutaneous nerves.

Diagnosis.—The diagnosis of leiomyomas occurring in the uterus is easily made. Leiomyomas developing in other organs are so rare that if a slowly growing tumor is found, the diagnosis of a fibroma, or, if rapidly growing, of a sarcoma is usually made. Only when it can be demonstrated that a large round abdominal tumor is connected with the stomach, intestinal, or bladder wall, should a myoma be thought of. Myomas of the skin may easily be mistaken for fibromas, as they are so painful. In these cases a myoma should be thought of if the changes so frequently associated with fibromas of the nerves (pigmented nævi and soft warts) are wanting.

Treatment.—An attempt should be made in the treatment to remove the tumor completely. If the tumors are scattered throughout the uterus, a hysterectomy should be performed. Encapsulated tumors may be enucleated. If the tumor is not encapsulated the dissection should be free, removing the muscle from which the tumor develops.

RHABDOMYOMAS

The rhabdomyomas are rare tumors which are composed of striated muscle fibers and a vascular intercellular connective tissue. Sometimes these tumors are benign, sometimes malignant. If other varieties of tissue are found in the growth they are classified as mixed tumors.

Gross Appearance and Histology.—Macroscopically they appear as nodular, usually well-defined, growths of varying consistency. They may attain considerable size. The interlacing of the fibers cannot be so easily seen in the grayish-red or gray tissue as in leiomyomas.

Microscopically no completely developed striated muscle fibers are found, but embryonal types of cells and fibers in different stages of development. The latter appear as hollow, tubular, or solid, multinucleated

bands of considerable length and different thicknesses in which both the transverse and longitudinal striations may be distinctly seen. The cellular forms contain spindle cells with long threadlike processes, parts of which have transverse striations, and irregular round or oval cells, often of considerable size, with one or many nuclei. All of these forms of cells frequently contain glycogen (Marchand), which may be distinctly seen, upon the addition of tincture of iodin, as large brown globules. On the fibers with transverse striation there may be indications of a poorly developed sarcolemma.

The structure of different rhabdomyomas differs, as in some cases the round or spindle cells predominate, in other cases the muscle fibers. The more the cells and fibers are grouped and interlaced, the more these tumors resemble histologically leiomyomas and fibromas.

Origin of Rhabdomyomas.—In discussing the origin of rhabdomyomas it is important to note that many mixed tumors, which undoubtedly

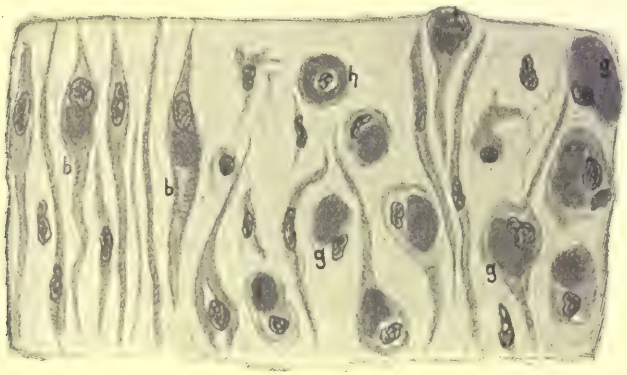


FIG. 390.—RHABDOMYOMA OF THE TEMPORAL REGION. *b, b,* Muscle cells cut parallel to their long axes in which fusiform enlargements and transverse striations may be seen. *g, g,* Round cells with processes; *h,* round cell without processes. The dark deposits represent drops of glycogen. (From Ribbert.)

are the result of developmental disturbances, contain striated muscle fibers, and that only the embryonal forms of these fibers are found in pure myomas. Rhabdomyomas are of congenital origin or develop in early life, and as they occur in organs which do not contain striated fibers, one seems justified in concluding that these tumors develop from germinal muscle tissue which was separated from normal physiological connections and displaced during embryonal life (Ribbert). It has also been suggested that the smooth muscle fibers composing a leiomyoma become transformed into striated fibers, thus forming a rhabdomyoma, and that normal, fully developed muscle might proliferate to form this type of tumor. There are a number of objections which may be raised against both of these suggestions (Borst and Ribbert).

Most Common Sites for Development.—Rhabdomyomas occur most frequently in the kidney, which is gradually destroyed as the tumor enlarges, so that finally a few remnants only are left. The tumor may reach the size of a child's head and send out nodular and polypoid processes into the pelvis of the kidney. Occasionally rhabdomyomas appear as polypoid tumors in the pelvis of the kidney, in the bladder, and as nodular growths in the testicles. Of the other organs the uterus and heart are the most important sites for the development of these tumors. Rhabdomyomas of the uterus appear as polypoid growths projecting into the vagina. They occur in the heart as congenital, multiple, usually small grayish-red nodules. Single tumors have been described in a number of different parts—in the œsophagus, stomach, parotid gland, prostate, in the muscles of the extremities, and about the buttocks and hips, in the tongue, orbit, temporal regions, etc.

Mode of Growth.—According to Ribbert, these tumors enlarge as the result of the proliferation of the young spindle and round cells, which later become transformed into striated muscle fibers. The growth is frequently expansive and slow, but it may be infiltrating in character. Then the tumor enlarges rapidly, the surrounding structures are invaded and metastases form. It is not known in these cases whether the tumor is to be regarded as a pure malignant rhabdomyoma or as a fibrosarcoma containing striated muscle fibers. The latter is frequently the case in mixed tumors, but it should not be forgotten that the young, non-striated, muscular elements may be easily mistaken for sarcoma cells.

Diagnosis.—The clinical peculiarities of these tumors are not sufficiently marked to enable one to make a positive diagnosis. The diagnosis is limited to determining whether the growth is benign or malignant, and then, depending upon the form, position, and rapidity of growth of the tumor, one can make a tentative diagnosis of a fibroma or of sarcoma, or, if it occurs in the genital tract, of a mixed tumor.

The operation which should be performed depends upon the extent, position, and character of the tumor.

LITERATURE.—*Becker.* Beitrag zur Kenntnis der wahren Muskelgeschwülste des Hodens. Virchows Arch., Bd. 163, 1901, p. 244.—*Büttner.* Ein Fall von Myom der weiblichen Urethra. Zeitschr. f. Geburtshilfe, Bd. 28, 1894, p. 136.—*Cohen.* Beiträge zur Histologie und Histogenese der Myome des Uterus und des Magens. *Ibid.*, Bd. 158, 1899, p. 524.—*Fujinama.* Ein Rhabdomyosarkom mit hyaliner Degeneration (Zylindrom) im willkürlichen Muskel. *Ibid.*, Bd. 160, 1900, p. 203.—*Hess.* Ein Fall von multiplen Dermatomyomen an der Nase. *Ibid.*, Bd. 120, 1890, p. 321.—*Lexer.* Myome des Mastdarmes. Arch. f. klin. Chir., Bd. 68, 1902, S. 241;—Myosarkom der Blase. Zentralbl. f. Chir., 1904, p. 22.—*Marchand.* Ueber einen Fall von Myosarcoma striocellulare der Niere. Virchows Arch., Bd. 73, 1878, p. 289;—Ueber eine Geschwulst aus quergestreiften Muskelfasern mit ungewöhnlichem Gehalt an Glykogen.

C. TUMORS COMPOSED OF NERVE ELEMENTS

CHAPTER I

NEUROMAS

Fibromas, myxomas, and sarcomas of nerves, which have also been called *false neuromas*, should not be classified with this group of tumors. Only those tumors composed of nervous elements belong here. They are exceedingly rare, developing apparently only from the sympathetic nerves upon which they appear as round or nodular growths resembling fibromas. Sometimes they attain considerable size, becoming as large as a man's head.

Histology.—They are composed of interlacing bundles of nerve fibers, the majority of which are non-medullated, the smaller part medullated. Between these fibers are found varying numbers of ganglion cells, which sometimes appear as poorly developed, round cells, at other times they give off axis cylinders. Neurilemma, interfibrillar connective tissue, and a few vessels are also found in these tumors. The terms *ganglio-neuroma* or *neuroma ganglio-cellulare* have been applied to these tumors, indicating that they contain both nerve fibers and ganglion cells.

The few cases (Knauss, M. B. Schmidt, Beneke, and Kredel) that have been observed have occurred mostly in small children. They appear as large tumors developing in the place of or near the sympathetic ganglia, or as small, multiple tumors of the skin developing supposedly from the sympathetic nerves, which contain a few ganglion cells, supplying the blood vessels (Knauss). The retroperitoneal tumor described by Beneke was benign, and caused symptoms only by its position and size.

Symptoms, Diagnosis, and Treatment.—The clinical peculiarities of these tumors are not striking enough to enable one to make a positive diagnosis.

Nothing definite is known concerning the origin of ganglio-neuromas. Their early, even congenital, occurrence and the presence of incompletely developed nervous elements indicate that they are the result of some disturbance in the development of the sympathetic nervous system (Ribbert, Borst).

Surgical treatment should be instituted when these tumors are accessible.

Amputation Neuromas.—The small nodular thickenings which develop upon injured nerves are called *traumatic neuromas*, although they are not true tumors, strictly speaking.

They are found especially upon the nerves in amputation stumps (so-called amputation neuromas), upon the central end of completely divided nerves or the sides of incompletely divided ones. They develop as the result of mechanical irritation of the nerves which lie immediately beneath the skin, that become caught in a scar or lie upon the edge of a bone (e. g., along the jaw and supraorbital ridge after saber cuts). There is a proliferation of the connective tissues of the nerve and a regeneration of medullated and non-medullated nerve fibers, which grow out for a short distance and then bend back to interlace with other proliferating fibers. The nodules, which form, never exceed in size twice the diameter of the nerve involved, and merely represent an excessive regenerative growth of the injured nerve.

Diagnosis and Treatment.—The diagnosis can be easily made. Small, hard nodules, which are painful upon pressure, lie beneath the skin in connection with the scar or immediately adjacent to it.

They may be easily removed. Precaution should be taken against their development when amputations are performed. The nerves to be divided should be drawn out from the wound and cut short so that they may retract some distance beyond the cut surfaces.

LITERATURE.—*Beneke.* Ueber gangliöse Neurome. Zieglers Beiträge zur path. Anatomie, Bd. 30, 1901, p. 1.—*Beneke* und *Kredel.* Ueber Ganglionneurome und andere Geschwülste des peripheren Nervensystems. Deutsche Zeitschr. f. Chir., Bd. 67, 1902, p. 239.—*Busse.* Ein grosses Neuroma gangliocellulare des Nervus sympathicus. Virchows Archiv, Bd. 151, 1898, p. 66 of the Supplement.—*Goldmann.* Beitrag zur Lehre von den Neuromen. Beitr. z. klin. Chir., Bd. 10, 1893, p. 13.—*Knauss.* Zur Kenntnis der echten Neurome. Virchows Arch., Bd. 153, 1898, p. 29.—*M. B. Schmidt.* Ueber ein ganglienzellenhaltiges wahres Neurom des Sympathikus. Virchows Archiv, Bd. 155, 1899, p. 557.

CHAPTER II

GLIOMAS

TUMORS developing from neuroglia, the stroma of the central nervous system, are called gliomas. They are composed of glia cells varying in their degree of development. They occur in the brain, spinal cord, and the eye, the last having histological peculiarities.

Peculiarities of Gliomas.—Gliomas of the brain occur as tumors, varying in size from a cherry to a fist, within the white and gray substance.

At times they infiltrate an entire hemisphere, the tumor gradually fusing with the surrounding tissues. It is frequently impossible to determine the boundaries of the tumor, as it may be of about the same color and consistency as the surrounding nervous tissue. Frequently such a tumor can be recognized macroscopically by the flattening of the convolutions covering it, or upon section by small, scattered, hæmorrhagic foci, necrotic areas or cavities resulting from softening and liquefaction. Gliomas occur most frequently in early childhood. Sometimes they appear as multiple, small, hard nodules upon the inner surface of the ventricle. They usually grow very slowly. The cellular forms grow rapidly, infiltrate and destroy the surrounding tissues; only rarely are the latter displaced by the growth. Gliomas do not develop above the surface of the brain.

Symptoms.—The symptoms are those of a brain tumor, and depend upon the position of the growth and the increase in intracranial pressure. Large hæmorrhages into the tumor are common, and apoplectiform seizures are frequent.

Varieties.—*Gliomas of the Spinal Cord.*—Gliomas of the spinal cord are most common in childhood. They frequently surround the central canal. They grow slowly, forming long, conelike growths, or transform a considerable extent of the cord into a gray mass, so that upon section only a narrow peripheral layer of normal tissue can be seen. Occasionally the growth extends through this peripheral layer and reaches the pia mater (Pels-Leusden). Cyst formation is frequent in gliomas of the cord. This is partly the result of dilatation of the central canal, and partly of softening of the tumor tissue. The symptoms are those of a spinal tumor. When cyst formation is marked, the symptoms may resemble those of syringomyelia.

The *prognosis* of gliomas of the brain and spinal cord depends upon their position. They have an infiltrating growth, and are therefore closely related to malignant growths, although they grow slowly and seldom form metastases.

Gliomas of the Eye.—Gliomas of the eye develop from the retina. They appear as nodular gray and white tumors, and grow into the vitreous humor. They grow rapidly, extending through the cornea externally, through the sclera into the orbit, or pass along the optic nerve to the cranial cavity. These tumors occur in children, not infrequently being bilateral. Some are of congenital origin. They destroy the eye affected, and frequently recur after enucleation of the eye. They are as malignant as sarcomas.

Histology.—Histologically, gliomas of the brain and spinal cord are composed of glia cells and a thick network of interlacing fibrillæ, part of which are the processes of the glia cells. The fewer the cells in

proportion to the number of fibrillæ, the harder the tumor. The number of blood vessels in these growths varies. Small or large, round or slitlike cavities lined with columnar cells are occasionally found. The latter are derivatives of the ependymal epithelium from which the glia cells develop. If proliferating ganglion and nerve cells are also present, the tumor is called a neuroglioma ganglionare (Ziegler). If most of the cells are fusiform in shape and the fibrillæ are not very pronounced, the tumor might easily be mistaken for a sarcoma.

Gliomas of the eye contain large numbers of round or oval cells with processes, and only occasionally an intercellular fibrillar substance. They also frequently contain cylindrical cells, radially arranged to form a lumen producing *rosettelike formations*. It has been suggested that these tumors develop from the neuroepithelium of the external layer of the retina (therefore they are called neuroepitheliomas by Wintersteiner).

Origin of Gliomas.—Probably all gliomas must be regarded as the result of some disturbances in embryological development of the brain, spinal cord, or eye. Congenital occurrence, development in early age, association with malformation of the brain and spinal cord, the bilateral occurrence of glioma of the eye, and their development in many members of the same family support this theory (Ribbert).

Diagnosis and Treatment.—A positive diagnosis of a glioma can be made when both eyes are affected. In other cases a glioma may be easily confused with a sarcoma. Usually one must be content in making a diagnosis of a tumor without being more specific.

Treatment is successful only in gliomas of the eye, and in these cases the contents of the orbit should be removed early. Gliomas of the brain and spinal cord are very frequently so situated, or are so extensive that complete removal is impossible.

LITERATURE.—*L. Bruns.* Gehirntumoren. Enzyklop. Jahrb. von Eulenburg, Bd. 5, 1895, p. 159.—*Greeff.* Gliom. in Orths Lehrb. der path. Anat. Path. des Auges, p. 400.—*Pels-Leusden.* Ueber einen eigentümlichen Fall von Gliom des Rückenmarkes mit Uebergreifen auf die weichen Häute des Rückenmarkes und Gehirns. Zieglers Beitr. zur path. Anat., Bd. 23, 1898, p. 69.—*Saxer.* Ueber Syringomyelie. Zusammenfassendes Referat über die seit 1892 erschienenen Arbeiten. Zentralbl. für allg. Path. u. path. Anat., Bd. 9, 1898, pp. 6 and 49;—Ependymepithel, Gliome und epitheliale Geschwülste des Zentralnervensystems. Zieglers Beitr. zur path. Anat., Bd. 32, 1902, p. 276.—*Ströbe.* Ueber Entstehung und Bau der Gehirngliome. *Ibid.*, Bd. 18, 1895, p. 405.—*Wintersteiner.* Ueber das Neuroepithelioma retinae. Leipzig-Wien, 1897.

D. TUMORS DEVELOPING FROM EPITHELIUM

CHAPTER I

FIBROEPITHELIAL TUMORS

In fibroepithelial growths there is a proliferation of both the epithelium and connective tissue, although they may vary in their degree of development, while in carcinomas the connective tissues play a subordinate rôle, forming merely the framework of the tumor which supports the cells and blood vessels.

The structural relationship between epithelium and connective tissue in fibroepithelial tumors finds its prototype in normal tissue. In the group of papillomas the same relation exists between epithelium and connective tissue as in the skin and mucous membranes. The structure of an adenoma resembles that of a gland, and the structure of epithelial cysts resembles in a number of ways the structure of skin and mucous membrane.

(a) PAPILLOMAS

Papillomas occur upon the free surface of the skin and mucous membranes. They are usually small tumors, rarely becoming larger than a walnut or hen's egg, have an expansive growth, and result from a hyperplasia of the epithelium with a corresponding new growth of the connective tissue and blood vessels. Long, branched papillæ, covered from summit to base with epithelial masses, extend down into the connective-tissue framework of the tumors. If the papillæ are surrounded by connective tissue and are held together, the new growth resembles a round nodule or a wart. If the papillæ and their branches are separated, deep-fissured, blackberry-, grape-, and villouslike tumors are formed which may be attached to the skin or mucous membrane by a broad base or thin pedicle. Marked cornification of the epithelium produces another variety of papilloma of the skin. In benign tumors the proliferation never extends below the subepithelial tissues.

Clinical Appearance.—Papillomas appear as single or multiple tumors, often being closely grouped, with broad bases or short pedicles. As a rule, they grow slowly. Sometimes they bleed profusely after injuries in which the base or pedicle is torn. After incomplete removal (ligation of pedicle, cauterization with weak caustics), they may begin to enlarge and grow rapidly. Usually, after growing slowly for some time, they remain stationary.

Varieties.—*Papillomas of the Skin.*—The epithelium covering papillomas of the skin becomes cornified. They are, therefore, harder than papillomas of the mucous membranes. These tumors develop most frequently upon the scalp of old people, but they also occur in the axillary fossa, the inguinal regions, in the folds below the breasts, on the back and perineum, about the anus, and upon other parts of the body exposed to continual irritation by sweat, rubbing, and uncleanliness. About the temporal regions they may develop from seborrhœic patches (*vide* p. 940).

FIG. 391.—PAPILLOMA OF THE SKIN WHICH HAS BEEN PRESENT FOR THIRTY YEARS (MAN SIXTY-FOUR YEARS OF AGE).

FIG. 392.—SECTION OF THE PAPILLOMA REPRESENTED IN FIG. 391.

Pointed condylomas develop upon the external genitalia and the adjacent skin following irritation produced by a gonorrheal discharge. In form and structure they are papillomas. They are not, however, tumors strictly speaking, but are hyperplastic growths, developing upon a chronically inflamed area. They disappear when the cause is removed.

Papillomas of Mucous Membranes.—Papillomas of the squamous mucous membranes are found in the mouth (lips, cheek, soft palate, and tongue), often developing from a leukoplakia, in the pharynx, larynx, œsophagus, and vagina. They usually develop in old people. Papillomas of the larynx, however, are found most frequently in children and young people; occasionally they are congenital. They are usually multiple, are characterized by rapid growth, and recur after removal.

The mucous membrane of the bladder, more rarely that of the ureter and pelvis of the kidney, may be the seat of single or multiple papillary tumors, the so-called villous polypi. They are soft, pedunculated

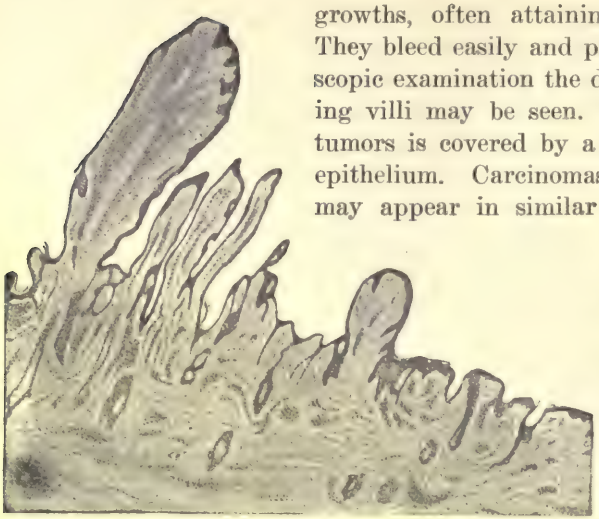


FIG. 393.—SECTION FROM THE BORDER OF A FISSURED PAPILLOMA OF THE SKIN.

growths, often attaining the size of a fist. They bleed easily and profusely. Upon cystoscopic examination the delicate, algælike, floating villi may be seen. The surface of these tumors is covered by a stratified, transitional epithelium. Carcinomas of the urinary tract may appear in similar forms (villous carcinomas) or develop from simple papillomas.

Papillary growths upon mucous membranes covered with columnar epithelium are rare, and are found almost only in the rectum. Occasionally they develop in the nose, uterus, stomach, and biliary passages. They are most common in old people, forming soft, villous tumors with a short pedicle or broad base, and vary in size from a pea to a hen's egg (villous polypi, tumor villosus). Injury by fæcal masses may be followed by profuse hæmorrhages.

Horny Warts and Cutaneous Horns. — Excessive cornification of the epithelium may lead to the formation of horny warts and cutaneous horns. The former appear as small, usually flat, nodules upon the skin. They are covered by a hard, fissured, horny layer of epithelium which holds the hypertrophied, threadlike papillæ together. Often the surface of these growths is fissured and of a thorny appearance, resembling a papilloma. These growths are found in both young and old people, occurring most fre-

FIG. 394.—MULTIPLE PAPILLOMAS OF THE LARYNX. (After P. von Bruns, Handbook of Practical Surgery.)

quently upon the fingers. They often disappear spontaneously or after a slight inflammation following an abrasion of the surface.

The cutaneous horn (cornu cutaneum) is most common in old people, and develops most frequently upon the hairy scalp and face (Figs. 396

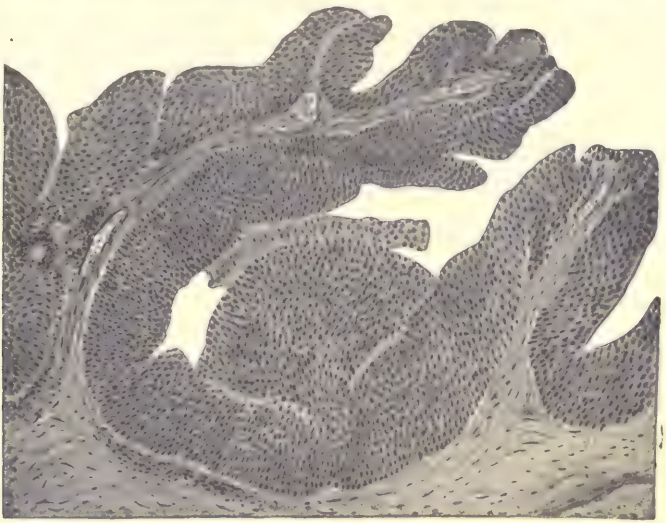


FIG. 395.—VILLOUS POLYP OF THE URINARY BLADDER. Pedunculated tumor the size of a fist, in a man sixty-five years of age.

to 398), the eyelids, nose, cheeks, lips, and ears being most commonly affected. Occasionally they are found upon the trunk, the extremities, the prepuce, and the scrotum. Multiple cutaneous horns are rare.

The new growth begins as a small wartlike nodule upon the skin, the

FIG. 396.—CUTANEOUS HORN ON THE EAR.

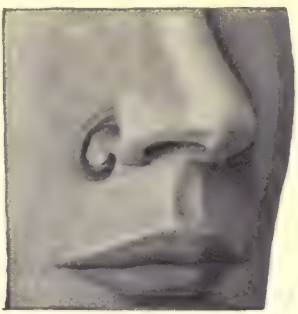


FIG. 397.—CUTANEOUS HORN ON THE NOSE.

horny layer of which proliferates to form a remarkable growth. The horns appear either as short, broad, conical projections, or as narrow but long (10 to 30 cm.), clawlike, crooked or twisted horns, the surfaces

of which are dark brown or black and have longitudinal striations. The skin at the base of such a horn is somewhat thickened and reddened. If a cutaneous horn is divided longitudinally, the hardest mass is found externally, and a soft, friable substance in the interior. Upon microscopical examination long, thin papillæ may be seen extending some

Fig. 398.—Cutaneous Horn of the Lip as seen from Without and Upon Section.

distance into the thickened epithelium. Proliferation of the papillary layer may be absent (Bätge). Occasionally a cutaneous horn develops from a papilloma or an atheroma.

The cutaneous horn always develops again after it falls off spontaneously, is rubbed off, is ligated off, or is incompletely removed.

Papillomas Occurring in the Brain.—In rare cases delicate, soft papillomas have been found in the ventricles of the brain. Such papillomas are connected with either the ependyma (Saxer) or the choroid plexus (Brüchanow).

Origin of Papillomas.—In discussing the origin of papillomas, it is important to remember that they are frequently secondary to chronic inflammatory conditions. Ribbert has been able to produce them experimentally by frequently injuring the same part of a rabbit's lip. Probably some papillomas are the result of developmental disturbances, especially the cutaneous papillomas and those tumors of the mucous membranes the epithelium of which does not correspond to the surrounding epithelium (the rare squamous-cell papillomas occurring upon mucous membranes covered with columnar epithelium).

Hæmorrhage from Papillomas and Symptoms.—Clinically, the greatest significance attaches to the hæmorrhage, which in the soft forms of papillomas may follow slight injuries and be very profuse. The other symptoms depend upon the position of the tumor. Papillomas of the larynx may cause dangerous dyspnœa and threaten life. Cutaneous horns and large papillomas of the eyelids and lips may cause ugly and distressing deformities (ectropion or drooping of the upper eyelid). Rapid enlargement, associated with induration of the base of the papilloma, indicates the beginning of a carcinoma which not infrequently develops upon such a growth.

Indications for Treatment.—The indication in the treatment of these growths is to remove the growth, together with the tissue from which it

develops. In small papillomas of the skin a number of applications of fuming nitric acid generally suffices. Similar growths on accessible mucous membranes should be cut away with scissors, and the wound then touched with the thermocautery. Large tumors and those with broad bases should be circumscribed and removed together with the tissue from which they spring. In the removal of papillomas of the larynx and bladder a preliminary operation is often performed when they are so large that they cannot be removed by intralaryngeal and intravesical instrumentation.

Incomplete removal is followed by rapid recurrence, and possibly favors the development of a carcinoma.

LITERATURE.—*Bätge.* Zur Kasuistik multipler Keratosen. Deutsche Zeitschr. f. Chir., Bd. 6, 1876, p. 474.—*Brüchanow.* Ueber einen Fall von Papillom des Plexus chorioideus ventriculi lateralis sin. bei einem 2½ jähr. Knaben. Prag. med. Wochenschr., Bd. 23, 1898, p. 585.—*Franke.* Ueber Hauthörner. Arch. f. klin. Chir., Bd. 34, 1887, p. 937.—*Mitvalsky.* Ein Beitrag zur Kenntnis der Hauthörner der Augenadnexa. Arch. f. Dermatol., Bd. 27, 1894, p. 47.—*Saxer.* Ependymepithel, Gliome und epithelial Geschwülste des Zentralnervensystemes. Zieglers Beitr. z. path. Anat., Bd. 32, 1900, papilläre Tumoren, p. 320.—*Spietschka.* Beitrag zur Histologie des Cornu cutaneum. Arch. f. Dermatol., Bd. 42, 1898, p. 39.

(b) ADENOMAS

Adenomas are fibroepithelial tumors in which the epithelial cells have a glandular arrangement. The finer structure of the tumor, the form of the cells, and their secretory function depend upon the character of the tissue from which the tumor springs.

Adenomas of the glands of the skin and mucous membranes differ from hyperplastic growths in that they do not reproduce the form and structure of the normal glands. Adenomas of the glandular organs differ from inflammatory hyperplasias in being encapsulated and in having no functional connection whatever with the surrounding tissues (Ribbert).

The majority of adenomas are benign growths. Some forms, however, invade the surrounding tissues and blood vessels, forming metastases.

Cystadenomas may be regarded as a variety of this class of tumors. Cystadenomas form when gland tubules become dilated by secretion poured out by the cells or when liquefaction, following regressive changes in the cells, occurs.

ADENOMAS OF THE SKIN

Adenomas of the skin are very rare and cannot always be easily differentiated from simple hyperplasias of the glands of the skin. A

carcinoma of the skin may develop upon an adenoma. It should be mentioned that if the adenoma is single and ulcerated the mistaken diagnosis of carcinoma may be made.

Adenomas of the sebaceous glands (adenomata sebacea) are most common upon the face, especially upon the nose and eyelids, occurring as single or multiple tumors. The tumors are soft and well encapsulated, vary in size from a pea to a walnut, have a warty or nodular form, and a red, slightly transparent or pearllike appearance. They lie within the skin, and the epithelial lobules composing the tumor, which resemble sebaceous glands, are imbedded in a connective-tissue stroma. These tumors are most common in old people, and grow slowly. At times they develop upon an old seborrhœic eczema and may ulcerate, after some superficial injury or as the result of regressive changes in the epithelium or hyaline degeneration of the connective tissue and blood vessels. They may become calcified (so-called calcified epithelioma) or transformed into a carcinoma (Barlow, von Noorden, Thorn).

They may easily be mistaken for endotheliomas, and can scarcely be differentiated from adenomas of the sweat glands. If ulcerated they may be mistaken for a flat carcinoma of the skin, especially if the skin surrounding the ulcer has become thickened and indurated as the result of inflammation.

They disappear after the use of strong caustics and the X-ray. In the treatment of larger tumors, excision is to be preferred because healing is much more rapid.

Adenomas of the sweat glands (adenomata sudoripara) are also rare. They occur as single, occasionally multiple, nodules of the skin and subcutaneous tissue. According to König, the multiple forms resemble lupus nodules. These tumors are most common in the face (forehead, temporal regions, nose, lips, chin) and in the scalp, but also occur in the skin of the breast and back, about the navel, in the inguinal regions, about the labia, and upon the extremities (*vide* Klauber).

They may develop in early childhood, but are most common in old people from fifty to sixty years of age. They grow slowly. The tumors developing in the skin appear as small nodules or pale sausagelike elevations of the skin, while the subcutaneous tumors are covered for a long time by normal skin, which, however, cannot be raised from the growth. Larger forms, the size of a plate or a child's head, have been observed (Klauber, Jupunoff). These larger growths, which are pedunculated and fungiform in shape, frequently become ulcerated and covered with crusts. The consistency of these growths varies, depending upon whether or not cysts develop. Chronic ulcers, from which flat, superficial carcinomas may develop, follow regressive changes in an adenoma.

Adenomas of the sweat glands consist of dilated and cystic gland

tubules lined with cylindrical epithelium, which may be arranged in the form of papillary growths. These tumors contain relatively large amounts of connective tissue, and are sharply defined against the surrounding tissues. They may occur in parts—for example, the upper lip—where normally there are no sweat glands, suggesting that some of these tumors arise from a congenital anlage.

The diagnosis is not simple. The tumors have no definite characteristics which enable one, when they are situated in the skin, to differentiate them from adenomas of the sebaceous glands, or if in the subcutaneous tissues, from lipomas or lymphangiomas. It is possible, even upon microscopic examination, to confuse them with the last and with endotheliomas. According to Borst, displaced germinal breast tissue should be considered in the diagnosis when the tumors occur in the skin covering the breast and in that of the back.

The same treatment as described in discussing adenomas of the sebaceous glands should be instituted for these tumors.

LITERATURE.—*Barlow.* Ueber Adenomata sebacea. Deutsch. Arch. f. klin. Med., Bd. 55, 1895, p. 61.—*Coenen.* Ueber Endotheliome d. Haut. Arch. f. klin. Chir., Bd. 76, 1905, p. 1100.—*Klauber.* Ueber Schweissdrüsentumoren. Beitr. z. klin. Chir., Bd. 41, 1904, p. 311.—*v. Noorden.* Das verkalte Epitheliom. Beitr. z. klin. Chir., Bd. 3, 1888, p. 467.—*Perthes.* Ueber gutartige Epitheliome wahrscheinlich kongenitalen Ursprunges. Deutsche Zeitschr. f. Chir., Bd. 65, 1903, p. 283.—*Thorn.* Ueber das verkalkte Epitheliom. Arch. f. klin. Chir., Bd. 56, 1898, p. 781.—*Stilling.* Einige Beobachtungen zur Anatomie und Pathologie des Lupus. Deutsche Zeitschr. f. Chir., Bd. 8, 1877, p. 72.

ADENOMAS OF THE MUCOUS MEMBRANE

The glands occurring in mucous membranes are usually involved in all the inflammatory growths affecting the latter, leading frequently, in the nose and its accessory sinuses and in the intestinal canal and urinary passages, to the formation of polypoid growths, the greater part of which is composed of loose connective tissue which has proliferated as the result of chronic inflammation. As adenomas of the mucous membranes occur most frequently as polyps, more rarely as flat tumors with broad bases, it is exceedingly difficult to differentiate between a true and an inflammatory new growth, or, in other words, between an adenoma of the mucous membrane and a glandular hyperplasia. The finding of glands in the polyps which do not correspond in form to the glands normally occurring in the part—for example, the occurrence of branched tubules in a polyp composed of Lieberkühn's glands—is indicative of an adenoma.

Clinical Characteristics of Adenomas of Mucous Membranes.—Adenomas of the mucous membranes are benign tumors which never invade the deeper tissues. The submucosa and muscularis mucosæ are the only

connective tissues involved in these growths. They differ in form and size. Their surfaces, which are covered by tissue resembling normal mucous membrane, are flat or somewhat nodular.

Hypertrophied and cystic mucous glands with watery contents are found in the connective-tissue stroma of the adenomas of the nasal mucous membrane, which occur as soft polyps or as pedunculated, grapelike tumors.

Adenomas of the Gastrointestinal Tract.—Adenomas of the gastrointestinal tract occur as single or multiple tumors. Frequently they appear in the form of polyps with thin pedicles, varying in size from that of a pea to that of a fist, with smooth, nodular or villous surfaces; more rarely as flat, circumscribed, soft thickenings of the mucous membrane.

FIG. 399.—SECTION FROM THE SUMMIT OF A RECTAL POLYP.

In the stomach they occur most frequently in the pyloric region, developing from the pyloric glands. If they are so situated and become large enough, they may obstruct the pyloric orifice and be diagnosed clinically as carcinoma.[1]

Adenomas of the intestinal canal develop from Lieberkühn's glands; those occurring in the duodenum from Brunner's glands (Salvioli). The single adenomas may occur in any part of the intestinal canal, but they are found most frequently in the lower part of the rectum, developing in children as well as in adults. The pedicle of such a tumor, which is usually round and never becomes larger than a walnut, may be stretched by the fæces passing over it or as the result of frequent prolapse until it becomes several centimeters in length.

Frequently hæmorrhage following stool or prolapse of the mucous membrane is the first symptom of a tumor of this character.

The presence of such a tumor may be easily determined by digital examination of the rectum.

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[1] Lexer once operated upon an adenoma of the stomach in an adult which was as large as a child's head. It was attached by a thick pedicle to the mucous membrane of the pylorus. The greater part of the tumor lay in the fundus.

After the pedicle is put on a stretch it should be ligated close to the mucous membrane and divided. If the polyp is torn during a bowel movement, the spurting vessel should be caught by a transfixion suture, or the bleeding point should be touched with the actual cautery.

Multiple Adenomas of the Intestines.—Multiple adenomas of the intestine give rise to a peculiar, often severe clinical picture. The mucous membrane of the rectum, which is chiefly affected, also that of the large intestine, and more rarely that of the small intestine immediately adjacent, is beset with numerous, closely set, small and large polypi (polyposis recti et intestini crassi). The symptoms of these multiple tumors, which occur in children and young adults, begin with an intestinal catarrh which resists treatment. The discharge of blood-stained masses of mucus is suggestive of multiple tumors of the intestinal mucous membrane. A history of heredity can frequently be elicited.

Profuse hæmorrhages occur in rapid succession, producing a marked anæmia. Occlusion of the bowel by the larger tumors, invagination of intestinal loops as the result of continual traction (e. g., invagination of the colon into the rectum), and finally the development of carcinoma (adenocarcinoma) are some of the complications. Twelve of the eighteen cases collected by Rotter died.

The *diagnosis* is made certain by an examination of the rectum.

Similar adenomatous polyps occur in the uterine mucous membrane and in young children about the umbilicus. The latter develop from remains of the vitelline duct.

These adenomas are to be regarded as the result of developmental disturbances, originating from pieces of mucous membrane which have been displaced and have become independent of the surrounding tissues.

Treatment.—In the treatment an attempt should be made to remove all the tumors which are accessible through the rectum. It will be necessary to perform a laparotomy in order to remove the adenomas situated in the higher intestinal loops. If the tumors have broad bases it will be necessary to circumscribe them and remove a portion of the wall of the stomach or intestine adjacent to them, closing the defect in the ordinary way. If the tumor has a long pedicle, all that is necessary after exposing the tumor is to ligate it close to the mucous membrane. In polyposis of the large intestine the results following even repeated operations are only temporary, because of the extent of the pathological changes.

LITERATURE.—*Port.* Multiple Polypenbildung im Tractus intestinalis. Deutsche Zeitschr. f. Chir., Bd. 42, 1896, p. 181.—*Rotter.* Verletzungen und Erkrankungen des Mastdarmes und des Afters. Handb. d. prakt. Chir., 2. Aufl., Bd. 3, p. 669.—*Schwab.* Ueber multiple Polypenwucherungen im Kolon und Rektum. Beitr. z. klin. Chir.,

Bd. 18, 1897, p. 353.—*Smoler.* Ueber Adenome des Dünn- und Dickdarmes. Beitr. z. klin. Chir., Bd. 36, 1902, p. 139.

ADENOMAS OF THE GLANDULAR ORGANS

Adenomas of the Salivary Glands.—These tumors are usually benign, being circumscribed and encapsulated. Sometimes they invade blood vessels and form metastases, in this way resembling clinically a malignant growth, although they resemble closely the structure of the organ from which they develop and would be classified histologically as benign tumors. The relative amounts of epithelial and connective tissues differ in the different tumors, and therefore the histological pictures of different adenomas vary a great deal. If the connective tissue predominates, the tumor is hard and of about the same consistency throughout, resembling a fibroma; therefore the term fibroadenoma. If the connective tissue is very cellular, the tumor is spoken of as an adenosarcoma; if mucoid, as an adenomyxofibroma (cystosarcoma phyllodes, myxofibroma intracanaliculare). The epithelial cells, which are usually arranged in a single layer, may be cylindrical, cubical, flat, or very irregular, and may form tubules which resemble ducts (tubular adenoma) or alveoli (alveolar adenoma). If the tubules or alveoli become transformed into cysts, a cystadenoma develops. If branched papillary growths develop in the tubules or cysts, an adenoma or cystadenoma papilliferum (papilloma, fibroma intracanaliculare) is formed.

Fig. 400.—Cystic Adenoma of the Parotid Gland.

Adenomas of the Mammary Gland.—Adenomas of the mammary gland appear as round, slowly growing nodules of different sizes and consistency. They may occur as multiple tumors in one or both breasts, are well encapsulated and benign, but recur after incomplete removal from pieces which are left behind. Because of their encapsulation they can be easily differentiated from diffuse hyperplasia of the breast. Adenomas of the breast are frequent in young women, but extremely rare in men. They may attain considerable size, especially the cystic forms. Finally, the skin covering

these tumors, which had been unchanged and perfectly movable, undergoes a pressure atrophy and becomes necrotic. The tumor is then exposed and develops above the level of the skin. The simple tubular and alveolar adenomas are hard, rich in connective tissue, and upon section have a homogeneous, grayish red color (fibroadenomas). If they develop from large tubules resembling ducts of the gland, large tortuous spaces and cysts form, which can be seen upon section. The tumor is then spoken of as a cystadenoma. If connective-tissue processes, carrying the epithelium with them, develop into the spaces, large cysts filled with pedunculated, papillary, villous, or grapelike growths are formed (cystadenoma papillare, papilliferum, or, depending upon the character of the stroma, fibroma or myxoma intracanaliculare, sarcoma arborescens, phyllodes).

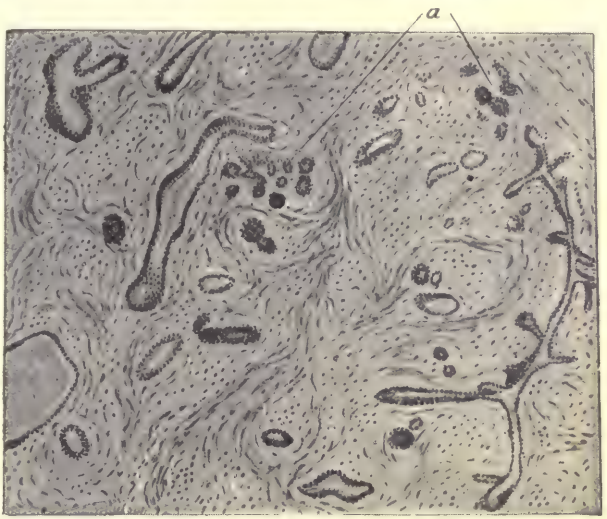


FIG. 401.—CYSTADENOMA OF THE MAMMARY GLAND. (*a*, ALVEOLAR ARRANGEMENT OF EPITHELIUM, AT OTHER POINTS A TUBULAR ARRANGEMENT.)

Mastitis Chronica Cystica.—A disease of the breast occurring frequently, especially in old women, and characterized by the formation of multiple cysts in one or both breasts, is regarded by Schimmelbusch as multiple cystadenomas; by König, because of its clinical course, as a chronic inflammation (mastitis chronica cystica). Upon section, small and large, brownish cysts are seen in the tissue of the breast, which is usually enlarged.

Adenomas of the Thyroid Gland.—Adenomas of the thyroid gland are nodular and circumscribed, differing from the diffuse or local hyperplasias which occur in the majority of goiters. The adenomas occurring in this gland gradually enlarge and may become as large as an apple. They may be congenital and are frequently multiple. Occasionally an adenoma of the thyroid becomes malignant, producing a pressure atrophy of the wall of a vein and invading its lumen. Cells are then carried by the blood stream and develop in different viscera and tissues. Slowly developing, secondary nodules then appear in the lungs and

bones where the cells find conditions most favorable for development. Gradually the bone involved undergoes pressure atrophy and spontaneous fractures may occur. [It has not been satisfactorily explained why adenomas and carcinomas of the thyroid gland are followed so frequently by the formation of secondary growths in bone. It should be remembered in this connection that carcinomas of the breast and prostate and hypernephromas are also frequently associated with secondary deposits in bone.]

Hypernephromas.—Adenomas of the adrenal glands (strumæ suprarenales, hypernephromas) appear as circumscribed nodules, the character of which may be easily recognized by the bright yellow color. These tumors, resembling histologically the cortex of the adrenal glands, develop from misplaced adrenal tissue. They occur in the adrenal gland and kidney (being probably the most frequent tumor occurring in the latter organ); more rarely in the broad ligament, in the epididymis, and on the under surface of the liver. Developing from adrenal rests lying in the cortex, they may form large tumors in the kidney and destroy almost all of the normal kidney tissue. If they rupture through their capsule, these tumors invade the renal vein and lead to the formation of extensive metastases.

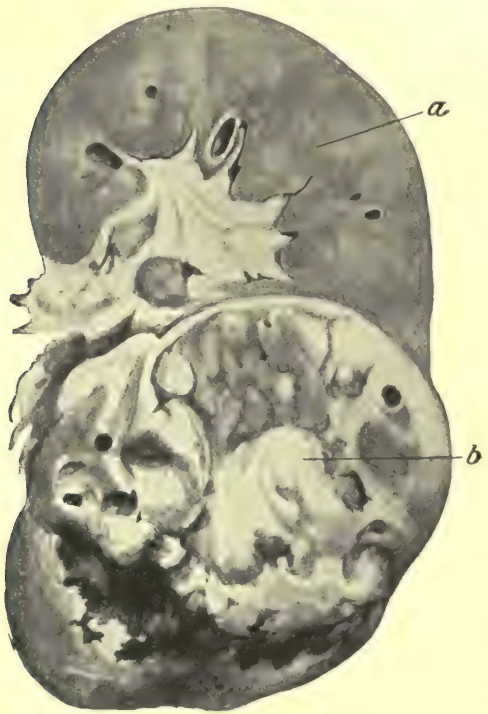


FIG. 402.—MALIGNANT HYPERNEPHROMA OF THE KIDNEY. (Woman thirty-five years of age.) *a*, Upper half of kidney not involved by the growth; *b*, tumor.

Adenomas of the Kidney.—Adenomas of the kidney occur as single or multiple encapsulated grayish white tumors which rarely become malignant. They never become larger than a cherry, and are composed either of tubules or cysts filled with papillary ingrowths.

Adenomas of the Liver.—Adenomas of the liver are composed of solid or hollow cell columns. They occur as round, soft, light brown nodules, or at times as large pedunculated tumors in the margin of the liver (von

Bergmann). These tumors are uncommon. Many have a tendency to an infiltrating and malignant growth, as they invade the radicles of the portal and hepatic veins and form metastatic growths. According to Ribbert, the development of multiple tumors which are histologically

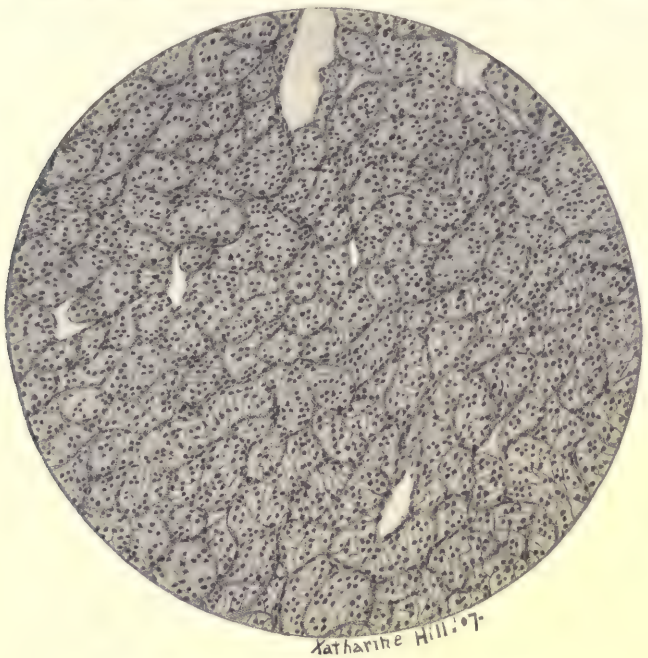


FIG. 403.—SECTION FROM A HYPERNEPHROMA.
(From Professor Bevan's Surgical Clinic.)

adenomas is best explained in this way. In spite of the tendency of these tumors to invade the vessels, metastases (in the lungs, lymph nodes, and bones) are relatively rare.

ORIGIN OF ADENOMAS

Adenomas develop from germinal tissue which has been displaced and encapsulated in the parenchyma of the organs in which they develop, forming independent centers of growth. The hypernephroma, which develops from adrenal tissue displaced into the cortex of the kidney, is the most striking example of this. The diagnosis and treatment of adenomas of the different organs belong to special surgery.

The cystic growths which occur in a number of organs and are probably due to developmental disturbances should be mentioned before closing the chapter upon adenomas.

The cysts or cystadenomas which occur so frequently in the ovaries should be mentioned first. These growths appear as large unilocular

or multilocular cysts in one or both ovaries. The cysts are lined with cylindrical epithelial cells which in some cases have proliferated to form papillary growths. The epithelium may have a glandlike arrangement. If the villous or papillary growths break through the cyst wall, the peritoneum becomes involved and the pelvis and the lower part of the abdominal cavity may become filled with papillary growths.

In congenital cystic disease of the kidney and liver these viscera are completely filled with small and large cysts (multilocular cystadenomas).

LITERATURE.—*Beer.* Ueber Nebennierenkeime in der Leber. Zeitschr. f. Heilkunde, Bd. 25, 1904, p. 381.—*v. Bergmann.* Zur Kasuistik der Leberchirurgie. Chir.-Kongr. Verhandl., 1893, II, p. 218.—*Buday.* Beiträge z. Zystenbildung in den suprarenalen Nierengeschwülsten. Zieglers Beitr. z. path. Anat., Bd. 24, 1898, p. 501.—*Dobbertin.* Beitrag z. Kasuistik der Geschwülste. *Ibid.*, Bd. 28, 1900, p. 42.—*Gierke.* Ueber Knochentumoren mit Schilddrüsenbau. Virchow's Arch., Bd. 170, 1902, p. 464.—*Goebel.* Ueber eine Geschwulst von schilddrüsenartigem Bau im Femur. Deutsche Zeitschr. f. Chir., Bd. 47, 1898, p. 348.—*Hildebrand.* Beitrag zur Nierenchirurgie. Deutsche Zeitschr. f. Chir., Bd. 40, 1895, p. 90;—Weiterer Beitrag zur pathol. Anatomie der Nierengeschwülste. Arch. f. klin. Chir., Bd. 48, 1894, p. 343;—Ueber den Bau gewisser Nierentumoren, ihre Beziehung zu den Nierenadenomen und zur Nebenniere, nebst Mitteilungen, über den Glykogenbefund in diesen, sowie in anderen Geschwülsten. Arch. f. klin. Chir., Bd. 47, 1894, p. 225.—*Holst.* Ueber doppelseitige primäre Nebennierentumoren. I.-D., Leipzig, 1904.—*Morris Wolff.* Beitrag zur Kenntnis der Tumoren der Mamma. I.-D., Rostock, 1899.—*Rehberg.* Untersuchungen über die Adenome der Nieren und ihre Entwicklung. I.-D., Freiburg, 1902.—*Sasse.* Ueber Zysten und zystische Tumoren der Mamma. Arch. f. klin. Chir., Bd. 54, 1897, p. 1.—*Schimmelbusch.* Das Fibroadenom der Mamma. *Ibid.*, Bd. 44, 1892, p. 102;—Das Zystadenom der Mamma. *Ibid.*, Bd. 44, 1892, p. 117.—*Sudeck.* Ueber die Struktur der Nierenadenome. Ihre Stellung zu den Strumae suprarenales aberratae. Virchows Arch., Bd. 133, 1893, p. 405;—Zur Lehre von den aberrierten Nebennierengeschwülsten in der Niere. *Ibid.*, Bd. 136, 1894, p. 293.

(c) EPITHELIAL CYSTS

This group comprises tumors the majority of which develop in the beginning as cysts. They are composed of epithelium and connective tissue. According to Ribbert, they may be classified with the fibroepithelial tumors, although many are closely related to the mixed tumors.

DERMOIDS OR DERMOID CYSTS

These tumors usually occur singly, appearing in the first few years of life. They are spherical or hemispherical in shape, depending upon the tension and pressure exerted by the soft tissues covering them.

Wall Lining and Contents.—The walls of these cysts are fairly thick and resistant. The outer surface is smooth, while the inner surface is rough, of a whitish color and covered with numerous fine and short, or thick and long hairs. The inner layer of the cyst wall is quite like

skin, as it contains epidermis, papillæ, hair, sebaceous and sweat glands. The outer layer, consisting of firm connective tissue, the inner part of which forms the connective tissue of the papillæ, is loosely attached to the surrounding tissue, and therefore the majority of these cysts can be easily enucleated without rupturing the wall. When occurring about the head, they may be firmly attached to the periosteum. The epithelial lining in many dermoids is incomplete. Where the lining is incomplete the epithelium is replaced by connective tissue rich in large polynuclear giant cells. The areas of different sizes, in which the epithelium is not present, are brown in color, smooth, and round (Fritz König). The giant cells frequently contain hair and fat crystals, and are to be regarded as foreign body giant cells. The contents of a dermoid cyst are a cheesy, yellowish-white mass, consisting of desquamated cells, the secretion of the sebaceous and sweat glands, numerous drops of fat, fat crystals, and cholesterin plates. Sometimes the contents are of a watery or serous character, or, after a hæmorrhage, of a reddish brown color.

Dermoid cysts occur in definite positions and are the result of developmental defects. They are found where, during embryonal life, there were clefts, furrows, or depressions of the surface of the body which later close, or where there were invaginations of the ectoderm. During the process of development cutaneous germinal tissue becomes buried beneath the skin and gradually develops to form cysts. Therefore these tumors usually occur in childhood and lie more frequently just beneath the skin than at a deeper level.

Dermoids of the Head and Neck.—Dermoids of the head and neck are the most common. Dermoids about the eye develop from pieces of ectoderm which are displaced during the invagination of the ectoderm to form the lens or during the fusion of the maxillary with the nasofrontal process. They occur most commonly: (1) Along the supraorbital ridge, at the outer canthus of the eye, about the glabella, at the root of the nose, or at the outer extremity of the orbital ridge; (2) within the orbit. The dermoids occurring along the orbital ridge appear as round growths, varying in size from that of a pea to that of a walnut. Those occurring in the orbit produce an exophthalmos. They are covered by normal skin, which may be easily displaced over the tumor, and lie directly beneath the skin or beneath the galea aponeurotica, the frontal portion of the occipito-frontalis, or the orbicularis oculi. When dermoids are deeply situated they sometimes produce changes in the bones about the orbit or in the other skull bones over which they are situated. When the dermoid lies directly upon the bone, a shallow depression with raised edges may be formed. Sometimes the depression may extend through the bone, and then the tumor comes in contact with the dura mater or the contents of the orbit. Occasionally a dermoid lying

outside of the orbit communicates by a narrow process with another cyst lying within the orbit. These cysts, communicating with each other (Zwerschsackdermoide of Krönlein) are not formed by the fusion of two separate cysts, but during the development of the cranial and facial bones the preëxisting cyst is surrounded by the bone and a part of the cyst is constricted to form the intermediate portion between the two parts. When the bone is fully developed a part of the cyst lies without, a part within the orbit.

FIG. 404.—DERMOID CYST AT THE OUTER END OF THE SUPRA-ORBITAL RIDGE.

Dermoids also occur at other points in the head, where, during development, the ectoderm can be invaginated easily. They occur about the anterior and posterior fontanelles, about the ear, just in front of the tragus, or over the mastoid process (developing from the aural anlage and the first branchial cleft). They also occur, but very rarely, on the face, on the dorsum and about the point of the nose, developing from the median nasal furrow (von Bramann, Lannelongue), in the cheek, at the side of the alæ nasi, developing from the naso-orbital cleft (von Bramann), in the middle of the cheek, developing from the transverse cleft of the cheek between the maxillary and mandibular processes (Lannelongue, Verneuil, Lexer).

It is striking that dermoids occur much more frequently about some of the embryonal clefts than others. Dermoids never occur about those clefts, fusion of the edges of which is easily prevented giving rise to a number of different developmental disturbances, such as lateral labial fissure, harelip, or cleft palate. Von Bramann believes that the piece of ectoderm forming the dermoid anlage is most frequently constricted off about those fissures, which are closed very early by the fusion of their borders. At the time of the closing of these clefts the proamnion is

closely related to the head, and there is more chance for the displacement of pieces of ectoderm during the closure of the cleft or the invagination of the ectoderm to form the lens, caused by adhesions with the proamnion, than after formation of the true amnion. Dermoids do not develop about the clefts that close later for the reason above given.

Dermoids of the neck occur most frequently in the lateral regions just below the submaxillary glands. When they occur in this position they are covered by the platysma and reach from the inner border of the sterno-cleido-mastoid to the digastric muscle. When they attain considerable size they develop posteriorly beneath the sterno-cleido-mastoid muscle. Dermoids occurring in the position above mentioned develop from the second branchial cleft. More rarely these cysts lie in the median line of the neck directly beneath the skin, sometimes above, at other times below the larynx, or in the jugulum (fusion of the branchial clefts, sinus cervicalis). The dermoids occurring in the floor of the mouth usually lie between the genioglossus muscles, extend toward the tongue, and elevate the mucous membrane of the floor of the mouth. In rare cases these tumors, resulting from imperfect fusion of the halves of the upper branchial arches, form on the outer side of the myelohyoid muscle, and then they are covered only by the skin of the submental region.

Dermoids Occurring in Other Parts.—The following positions in which dermoids also occur should be mentioned: (1) Dermoids of the chest, situated in the anterior or posterior mediastinum, which, like those occurring in the abdominal wall about the navel or in the omentum and mesentery, are formed from pieces of ectoderm invaginated during the closure of the thorax and abdominal wall. (2) Dermoids of the pelvic connective tissues, of which those not connected with the ovary or constricted off from it develop from ectoderm invaginated from the perineum. (3) Cysts of the retroperitoneal connective tissue, which, like those occurring along the spermatic cord (Wrede), develop from the ectodermal remains of the Wolffian duct. (4) The extremely rare dermoids of the scrotum, penis, and raphe, which are formed during the formation of the perineum or the fusion of the anlage of the external genitalia. (5) Dermoid cysts occurring in the sacral or coccygeal regions, which are relatively frequent upon the dorsum of the sacrum and coccyx, and often become transformed as the result of infection or traumatism into fistulæ which resist treatment. [Some of these cysts appear as funnel-shaped invaginations of the skin, and, as they contain hair, they are called pilonidal cysts.] Dermoids occurring in front of the sacrum and coccyx are formed during the development of the perineum or the formation of the cloaca. (6) Intracranial dermoids are situated at the base of the brain between the dura mater and bone or

within the pia mater. Dermoids of the vertebral column, associated with spina bifida, and of the pharynx are extremely rare.

Dermoids of the testicle and ovary are rarely simple. The majority are complicated dermoids, cysts containing a number of different tissues, and should be classed with the teratoid tumors (*vide* p. 980).

Epidermoids.—In the histological examination of many dermoids it is found that the lining of the wall of the cyst is not composed of the cutis, but of many layers of flat epithelium containing papillæ and the stratum Malpighii, but no hair or sweat glands. These tumors are called epidermoids and are probably formed by the invagination of germinal cutaneous tissue containing no glandular or hair anlage. Perhaps the difference between dermoids and epidermoids depends upon the time at which the tissue was displaced. It is quite conceivable that the germinal tissue displaced early would be more apt to form fully developed skin than that displaced later. In the post-embryonal transplantation of skin epithelial cysts and not dermoids develop.

According to Frank (p. 989), some of the atheromas, those lying subcutaneously, belong to the class of epidermoids, as they develop from germinal tissue displaced during the formation of the hair follicles and sebaceous and sweat glands.

Suppuration and Fistula Formation.—The formation of a fistula following a trauma or inflammation is the most important of the changes which may occur in a dermoid. Fistula formation occurs most frequently in dermoids situated about the coccyx, occasionally in those situated about the nose. It is indicated by the protrusion of a small tuft of hair. Suppuration of the cyst follows infections secondary to injuries, occasionally hæmatogenous infections. In rare cases a carcinoma develops in the cyst wall (H. Wolff and others).

Diagnosis.—The diagnosis is easily made. It is based upon a number of characteristics common to dermoids. The position of the tumor and its early appearance, even before puberty, are of the greatest diagnostic significance. Besides, the cysts are sharply circumscribed and smooth, and but slightly adherent to the surrounding tissues, being, therefore, freely movable. They are covered by normal skin, which can be easily displaced over the tumor. Their consistency differs, depending upon their contents. Frequently an indistinct or decided fluctuation can be elicited; often these tumors have a doughy consistency.

Dermoids of the head and face can usually be easily differentiated from atheromas, which are attached to the skin at the point at which the duct escapes and therefore move with it. Cysts occurring at the root of the nose, about the glabella, and at the inner canthus of the eye may be easily confused with nasofrontal and nasoethmoidal encephaloceles, if they lie in a depression in the bone and give the im-

pression of communicating with the cranial cavity. In making the differential diagnosis it is important to note whether there are symptoms of intracranial pressure and whether the tumor decreases in size under pressure. Both of these characteristics are often prèsent in encephaloceles. It is scarcely possible to differentiate retrobulbar orbital dermoids from other varieties of tumors. Dermoids occurring in the lateral regions of the neck and the branchial cysts, which are genetically the same, may, in spite of the definite positions in which they occur, be confused with tuberculous abscesses and lipomas. Examination should be made for swollen adjacent lymph nodes, as these would be indicative of tuberculosis. The smooth surface of the cyst may prevent mistaking it for a lipoma, which is usually lobulated. Aspiration is uncertain. Dermoid cysts in the floor of the mouth usually have a median position, differing in this way from ranulæ, which usually are situated more to the sides of the floor of the mouth cavity.

Treatment.—Dermoid cysts should be enucleated. Enucleation is difficult only when the wall has been partially destroyed by suppuration. If during the removal of the cyst any of the wall is left, the tumor rapidly recurs. Simple incision, permitting of the escape of the contents of the cyst, is not sufficient. Only in very large retroperitoneal, thoracic, and sacrococcygeal cysts should one be content with this line of treatment, as a destruction of the sac cannot be expected after drainage.

LITERATURE.—*Aschoff.* Zysten. Lubarsch u. Ostertags Ergebnisse, 2. Jahrg. Wiesbaden, 1897, p. 456.—*v. Bramann.* Ueber die Dermoide der Nase. Arch. f. klin. Chir., Bd. 40, 1890, p. 101.—*Franke.* Die Epidermoide (sog. Epithelzysten). Deutsche Zeitschr. f. Chir., Bd. 40, 1895, p. 197;—Ueber das Atherom, besonders mit Bezug auf seine Entstehung (das Epidermoid). Arch. f. klin. Chir., Bd. 34, 1887, p. 507.—*Klapp.* Zur Kasuistik der Dermoide des Mundbodens. Beitr. z. klin. Chir., Bd. 19, 1897, p. 608.—*Fritz König.* Beiträge zur Anatomie der Dermoide und Atheromzysten. Arch. f. klin. Chir., Bd. 48, 1894, p. 164.—*Krönlein.* Dermoide der Orbita. Beitr. z. klin. Chir., Bd. 4, 1889, p. 149.—*Lannelongue* et *Achard.* Traité des kystes congénitaux. Paris, 1886.—*Lannelongue* et *Ménard.* Affections congénitales. Paris, 1891, T. 1.—*Längner.* Die angeborenen Geschwülste der Steissbeingegend und des Beckenbindegewebes. I.-D., Berlin, 1902.—*Lexer.* Ueber teratoide Geschwülste in der Bauchhöhle und deren Operation. Arch. f. klin. Chir., Bd. 61, 1900, p. 648.—*de Quervain.* Ueber die Dermoide des Beckenbindegewebes. Arch. f. klin. Chir., Bd. 57, 1898, p. 129.—*Sänger.* Dermoidzysten des Beckenbindegewebs. Arch. f. Gynäkol., Bd. 37, 1895, p. 100.—*Heinr. Wolff.* Karzinom auf dem Boden des Dermoids. Arch. f. klin. Chir., Bd. 62, 1900, p. 731.—*Wrede.* Die Dermoide des Samenstranges. Beitr. z. klin. Chir., Bd. 48, 1906, p. 273.

TRAUMATIC EPITHELIAL CYSTS

Not infrequently small, round cysts, which never become larger than a cherry, are found in the palm of the hand and upon the flexor sur-

faces of the fingers. These cysts, resting upon the palmar fascia or upon the sheaths of the flexor tendons, may be displaced quite easily. The skin, in which small scars indicating the nature and origin of the cyst may be seen, is slightly adherent to it. The walls of the cysts are composed of loose connective tissue, more or less firmly fused with surrounding structures, and are lined by squamous epithelium. The contents are similar to those found in dermoids.

Etiology.—These cysts develop from small pieces of skin which have been displaced by trauma—therefore have been called traumatic epithelial cells (Garrè)—or originate from appendages of the skin (hair follicles, sebaceous and sweat glands) which have been displaced and carried into the tissues by some penetrating foreign body (Pels-Leusden).

An epithelial cyst is represented in Fig. 405. It was situated in the palm of the hand and developed six months after a gunshot wound. A small, flattened, lead bullet is encapsulated in the subcutaneous tissues. The capsule does not surround the foreign body closely, being separated from it by a cheesy mass. Upon one side a piece of thickened epidermis (*a*), which was carried into the deeper tissues by the bullet, surrounds it like a hood. This epidermis forms part of the cyst wall.

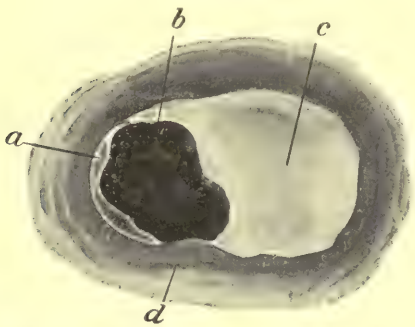


Fig. 405.—Traumatic Epithelial Cyst of the Palm of the Hand. *a*, Germinal cutaneous tissue; *b*, bullet; *c*, cheesy contents of cyst; *d*, connective-tissue capsule with epithelial lining.

Fig. 406.—Traumatic Epithelial Cyst of the Index Finger.

Wiemann has also demonstrated foreign bodies in two epithelial cysts.

Experimental Production.—Similar cysts may be produced experimentally. After a piece of skin has been transplanted into the subcutaneous tissues or into the abdominal cavity it becomes encapsulated by connective tissue, which, however, cannot become united with the

epithelial surface of the transplanted epidermis. A small space, which becomes filled and distended with desquamated epithelium, remains between the epithelium and connective tissue, and the walls of the space become lined with epithelium formed by the proliferation of the epithelium of the transplanted skin.

The treatment consists of complete extirpation.

LITERATURE.—*Garrè.* Ueber traumatische Epithelzysten der Finger. Beitr. z. klin. Chir., Bd. 11, 1894, p. 524.—*Pels-Leusden.* Ueber abnorme Epithelisierung und traumatische Epithelzysten. Deutsche med. Wochenschrift, 1905, p. 1578.—*Wegner.* Beitrag zur Lehre von den traumatischen Epithelzysten. Deutsche Zeitschr. f. Chir., Bd. 50, 1899, p. 201.—*Wiemann.* Epidermoide (Epithelzysten) mit Einschluss von Fremdkörpern. Zentralbl. f. Chir., 1902, p. 578.—*Wörz.* Ueber traumatische Epithelzysten. Beitr. z. klin Chir., Bd. 18, 1897, p. 753.

CHOLESTEATOMAS

Cholesteatomas are tumors which are closely related to dermoids and epidermoids. Their walls are alike, but their contents differ.

Contents.—The contents of these tumors consist of white, pearllike, glistening masses, which are concentrically arranged. These masses are dry and upon section become broken up into fine lamellæ. They consist of cornified, firmly compressed epidermal scales, and contain large amounts of fatty detritus and cholesterin; for this reason the term cholesteatoma has been applied to them. The walls are composed of a stratified epithelium. Transitional stages between the flattened epithelium and the cornified masses may be seen in any part of the tumor. Sebaceous and sweat glands and hair are but rarely found. External to the epithelium is a layer of loose connective tissue.

Clinical Course.—These tumors grow slowly without producing symptoms for a long time, and finally may become as large as a hen's egg or larger. If the wall undergoes necrosis as a result of the pressure of the contents, the latter extend along the spaces of the surrounding connective tissues. Finally even bone may undergo atrophy following pressure produced by these masses.

Most Common Sites for Development.—The most frequent and most important situations for cholesteatomas are the middle ear, the pia mater, and the urethra.

They develop most frequently in the tympanum and the antrum, varying in size from that of a cherry seed to that of a hen's egg. They may gradually produce a pressure atrophy of the bone and give rise to dangerous symptoms. These growths almost always cause, after a time, an otitis media and extend, when the bone is perforated, into the cranial cavity. Some of these tumors develop from ectoderm which has been displaced into the mastoid cells or the tympanum during the develop-

ment of the ear (von Mikulicz and Küster), and some follow chronic inflammations, the squamous epithelium of the external auditory meatus growing through the perforation of the membrana tympani to replace the cylindrical epithelium destroyed by suppuration. The secretion of the epithelium then becomes inspissated to form with the desquamated cells the cholesteatomatous masses (pseudocholesteatoma of von Tröltsch,

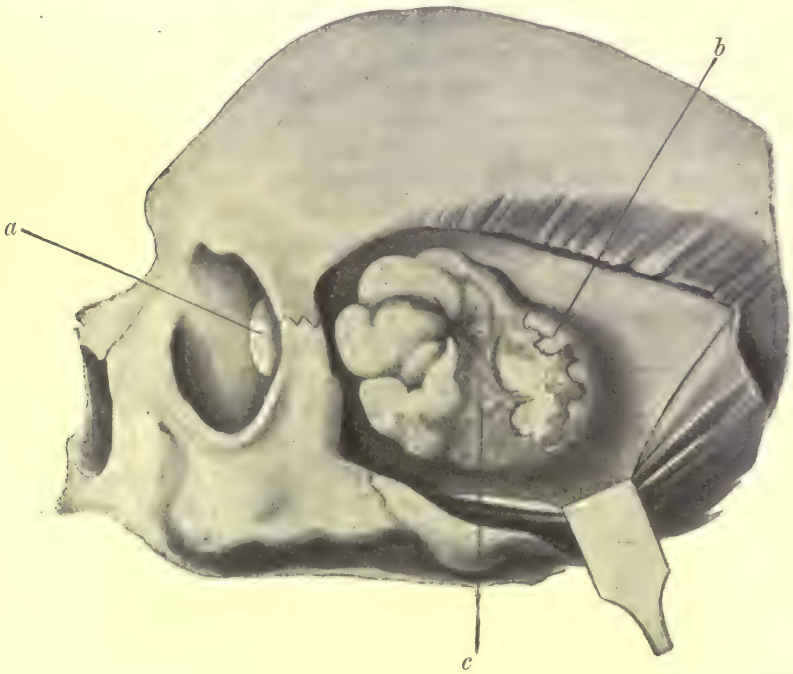


FIG. 407.—CHOLESTEATOMA OF THE SKULL BONES, WHICH HAS INVADED THE ORBIT. *a*, Part of tumor which has invaded the orbit; *b*, external table elevated by tumor mass; *c*, layer of bone, remains of external table.

Habermann, Körner). It is possible that the epithelium of the middle ear undergoes a metaplasia in some cases, for cases have been observed in which a perforation of the membrana tympani, permitting of the ingrowth of squamous epithelium, could be positively excluded (Borst). In chronic inflammations of the middle ear similar masses are found (otitis chronica desquamativa), but the epidermoidal sac is wanting in all these false cholesteatomas (Kümmel).

Cholesteatomas of the pia mater appear as round or nodular tumors, which grow very slowly. They usually occur at different points about the base of the brain; more rarely within the ventricles. The capsule of the tumor, which is lined by a number of layers of flattened epithelium, is fused with the pia mater. Boström regards these tumors as

epidermoids, and believes that they develop from germinal ectodermal tissue displaced into the anlage of the pia mater about the fourth or fifth week of embryonal life. Borst and others regard them as endotheliomas.

A few cases of cholesteatoma of the bones of the face and skull have been observed. They have been found in the frontal, occipital, and parietal bones (Blecher), in the temporal bone, the pterygoid plates of the sphenoid, in the bones of the orbit (Lexer), and in the mandible (von Mikulicz), appearing as slowly growing tumors which gradually destroyed the bone. Not infrequently these tumors are epidural. Borchardt has removed such a tumor from the posterior cranial fossa.

Cholesteatomas of the urinary passages are not true tumors, but the products of degeneration of flat epithelium, the occurrence of which in these passages is abnormal. These cornified masses occur in the urethra, back of strictures, in the bladder, the pelvis of the kidney, and in the ureter (Brüchanow).

Diagnosis.—A positive diagnosis of cholesteatoma can be made only when the characteristic masses are discharged. In making the diagnosis the symptoms produced by the pressure of the tumor should also be considered, but it is impossible to differentiate cholesteatomas from other tumors, unless the characteristic masses are discharged.

Treatment.—The treatment consists of removal of the cornified masses, and, in the true cholesteatomas, of the capsule also.

LITERATURE.—*Borchardt.* Cholesteatom der hinteren Schädelgrube. Chir.-Kongress Verhandl., 1905, II, p. 496.—*Boström.* Die pialen Epidermoide, Dermoide und Lipome und duralen Dermoide. Zentralbl. f. allg. Path., Bd. 8, 1897, p. 1.—*Brüchanow.* Ueber einen Fall von sog. Cholesteatombildung in der Harnblase. Prag. med. Wochenschr., Bd. 23, 1898, p. 525.—*Haug.* Ueber das Cholesteatom der Mittelohrräume. Zentralbl. f. allg. Path., Bd. 6, 1895, p. 124.—*Habermann* Zur Entstehung des Cholesteatoms des Mittelohrs. Arch. f. Ohrenheilkunde, Bd. 27, 1889, p. 42.—*Kümmel.* Die Verletzungen und chirurgischen Erkrankungen des Ohres. Handb. d. prakt. Chir., 2. Aufl., Bd. 1, p. 404.—*Unterberger.* Ein echtes Cholesteatom der Schädelknochen. Deutsche Zeitschr. f. Chir., Bd. 81, 1906, p. 90.

ADAMANTINOMAS AND FOLLICULAR CYSTS OF THE JAW

Adamantinomas are benign tumors of the jaw which occasionally occur in young people. They grow very slowly and may become as large as an apple or a fist. These tumors usually lie encapsulated within the bone and gradually expand the latter, so that finally the tumor is covered only by a shell of bone. If the tumor is cystic, a " parchment crackling " can be elicited when the thin shell of bone covering it is palpated. Adamantinomas of the maxilla may grow into the antrum of Highmore and completely fill it.

Histology.—Upon section these tumors differ very much. They appear either as homogeneous, yellow, resistant masses, the consistency of which is very much like that of a fibroma, or as small or large cysts (therefore the terms "Multilocular cystomas of the jaw, epithelioma adamantinosum cysticum"). The entire mass may be enucleated from the expanded bone, unless the latter has become so thinned that it is fractured during the enucleation (Fig. 408).

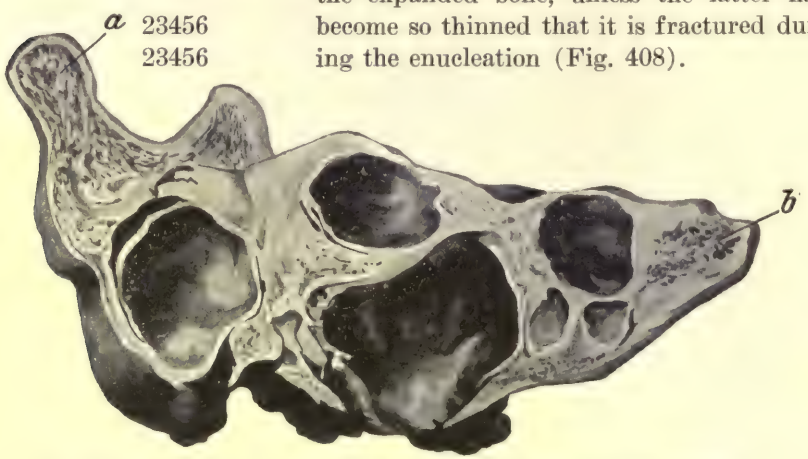


FIG. 408.—MULTILOCULAR CYSTOMA OF THE MANDIBLE (ADAMANTINOMA).
a, Condyle; *b*, symphysis.

Microscopically the solid tumors consist of a connective-tissue stroma throughout which are scattered interlacing, narrow, and wide columns of epithelial cells. If these are abundant the tumor is very similar, histologically, to a carcinoma, but differs from a carcinoma in that it is encapsulated. Cylindrical epithelial cells are found at the periphery of these columns, while the cells occupying the center are either flat, arranged concentrically, or are stellate and branched.

If the epithelial masses undergo regressive changes, small cavities form within the cell columns, which later enlarge. The cysts are usually lined with but a single layer of cylindrical epithelium, and resemble, histologically, cystadenomas. The histological picture is still different if papillary growths develop into the cavity or if the stroma between the columns of epithelial cells becomes ossified.

Origin of Adamantinomas.—The form and arrangement of the epithelium indicate that these tumors arise from the enamel organ; therefore the term adamantinoma. They develop from the remains of the epithelium of the enamel organ (débris paradentaires, Malassez), which lie about the teeth and can even be demonstrated under normal conditions. Perhaps the hyperæmia associated with inflammation may be the exciting cause of these growths.

Diagnosis.—It is difficult to make a positive diagnosis. Small, solid tumors may be mistaken for osteomas; cystic tumors for simple cysts of the jaw, or if it is not known that the tumor has existed for some time, for soft, central sarcomas.

Treatment.—The treatment consists of free exposure of the tumor and enucleation. The jaw should be resected in order to prevent recurrence if a cystic tumor has caused a pressure necrosis of the jaw and rendered it fragile (Fig. 408).

Follicular Cysts.—Follicular cysts of the jaw are simple cysts which develop from misplaced or supernumerary tooth buds. The position of these cysts varies. They may occur upon any part of the jaw (e. g., the ramus of the lower jaw, in the orbit).

They are found chiefly in young people. When they occur in adults they develop about the wisdom teeth only, and most frequently in the mandible. They grow slowly, without causing pain, producing a localized expansion of the bone. They are found more rarely in the maxilla, and then easily extend into the antrum.

These cysts have smooth walls, which are lined by an epithelial membrane derived from the tooth bud, and contain a rudimentary or fully developed tooth. The fluid contents of the cyst are seromucous in character and rich in cholesterin.

FIG. 409.—ADAMANTINOMA.

Follicular cysts are common, and the diagnosis is not so difficult. But it should be remembered that the thinned and expanded cortical layer of bone may conceal a central sarcoma or a cystic adamantinoma, and that the antrum of Highmore may be dilated as the result of chronic inflammation.

The indications for treatment are to remove the cortical layer of bone, to expose the cyst, and then to remove the cyst wall.

Periosteal Cysts.—Periosteal cysts of the jaws are to be differentiated from both of these forms of true cysts, above mentioned, which develop within the bone. Periosteal cysts are inflammatory growths. They frequently develop after a periostitis involving the root of the tooth. After

the destruction of the bone surrounding the diseased root, a cloudy, mucoid fluid containing cholesterin collects beneath the periosteum and produces a flat, fluctuating swelling on the outer surface of the jaw, which usually is situated about the molar or bicuspid teeth. Such a swelling may even rupture into the antrum of Highmore.

It is questionable whether these cysts are produced by inflammation alone. The finding of epithelium in the walls of these cysts (Charcot, Malassez) suggests that the displacement of epithelium has something to do with their development. It may be that displaced epithelium is essential to the formation of the cyst, and that the inflammation acts as the exciting cause.

Periosteal cysts are painful, grow quite rapidly, are situated just beneath the periosteum, and occur at any age. When these facts are taken into consideration, periosteal may be easily differentiated from central cysts.

Free incision permitting of the discharge of the fluid, curettage of the depressed area in the jaw, with removal of the diseased root, and subsequent tamponade, are followed by a permanent cure after some days.

If the cyst is large it is best to remove a large piece of the external wall.

LITERATURE.—*Becker.* Zur Lehre von den gutartigen zentralen Epithelialgeschwülsten der Kieferknochen. Arch. f. klin. Chir., Bd. 47, 1894, p. 52.—*E. Bennecke.* Beitrag zur Kenntnis der zentralen epithelialen Kiefergeschwülste. Deutsche Zeitschr. f. Chir., Bd. 42, 1896, p. 424.—*Goebel.* Ueber Kiefertumoren, deren Entstehung auf das Zahnsystem zurückzuführen ist. Sammelreferat. Zentralbl. f. Path., Bd. 8, 1897, p. 128.—*Haasler.* Die Histogenese der Kiefergeschwülste. Arch. f. klin. Chir., Bd. 53, 1896, p. 749.—*Kruse.* Ueber die Entwicklung zystischer Geschwülste im Unterkiefer. Virchow's Arch., Bd. 124, 1891, p. 137.—*Malassez.* Sur le rôle des débris épithéliaux paradentaires. Arch. de physiol., 1885.—*Nasse.* Paradentäres zentrales. Adenokystom des Unterkiefers. Chir.-Kongr. Verhandl., 1890, I, p. 129.

EPITHELIAL CYSTS DEVELOPING FROM NORMAL EMBRYONAL ANLAGE

Cysts may develop from persisting, noninvoluted remains of different embryonal fissures and canals of ectodermal and entodermal origin. The most important of these cysts occur in the neck, the floor of the mouth, and in the abdomen, the last developing from the urachus and the vitelline duct.

Branchial Cysts.—The epithelial cysts occurring in the neck are divided, depending upon their position and origin, into the lateral and median; the former developing from the branchial clefts, the latter from the thyreoglossal duct. These cysts are closely related genetically to the lateral and median cervical fistulæ.

Branchial cysts usually develop in the young. They may even be

congenital. They form painless swellings, which grow slowly. Often the beginning of the enlargement is not noted. They develop in the region between the jaw, the inner border of the sterno-cleido-mastoid muscle, and the hyoid bone. Externally they are covered by the platysma. They extend inward to the digastric muscle. They may become larger than a fist, and then they extend downward almost to the clavicle and backward beneath the sterno-cleido-mastoid muscle into the posterior triangle of the neck. The swelling produced by such a cyst is flat or hemispherical, has fairly well-defined boundaries, a smooth surface, and, depending upon the character of the contents, a doughy or fluctuating consistency. The skin covering it is normal and can be raised from it, but the tumor proper can be displaced but little, as it is adherent to the deeper tissues. The firm, smooth wall of the cyst has either a white lining like that of a dermoid, or a grayish red lining, which is often very granular, resembling mucous membrane. The contents are cheesy, mucoid, or serous, with a number of transitional forms. The symptoms are entirely dependent upon the size and position of the cyst.

Branchial cysts usually develop from remains of the second branchial cleft; exceptionally, from the first or third (Fritz König). Genetically they are closely related to congenital branchial fistulæ, the internal orifice of which usually communicates with the supratonsillar fossa, when the fistula is complete; the external orifice emptying upon the skin anterior to the sterno-cleido-mastoid muscle at any level of the neck.

The epithelial lining of these cysts, like that of the fistulæ, differs in character. The inner part of branchial fistulæ is of entodermal origin and is lined either with ciliated epithelium or with squamous epithelium provided with lymphadenoid tissue or follicles (like pharyngeal mucous membrane), while the outer part is derived from ectoderm, and is lined with squamous epithelium containing papillæ, and often glands of the skin. There are branchial fistulæ, however, which are lined throughout with cylindrical epithelium, having developed entirely from the entodermal portion of the cleft.

The lining and contents of branchial cysts differ, depending upon whether the epithelium from which the cysts develop is derived from the entodermal or ectodermal portion of the cleft. Cysts lined with skin or epidermis have cheesy contents, like a dermoid, and do not differ from dermoids or epidermoids (p. 916) of a doughy consistency. Cysts derived from the entoderm have seromucous contents and fluctuate distinctly. The inner surface of these cysts often resembles closely a granular tuberculous membrane, but the granulations are firm and cannot be wiped away as in the tuberculous membrane, for they are

produced by numerous lymphatic follicles situated just beneath the mucous membrane. Occasionally, different forms of epithelium are found in different parts of the cyst, or if the cysts are multilocular, the smaller compartments may be lined by different kinds of epithelium. The outer layers of the wall of the cyst may, like those of a branchial fistula, contain lymphadenoid tissue, striated muscle fibers, or cartilage (*vide* Plate II).

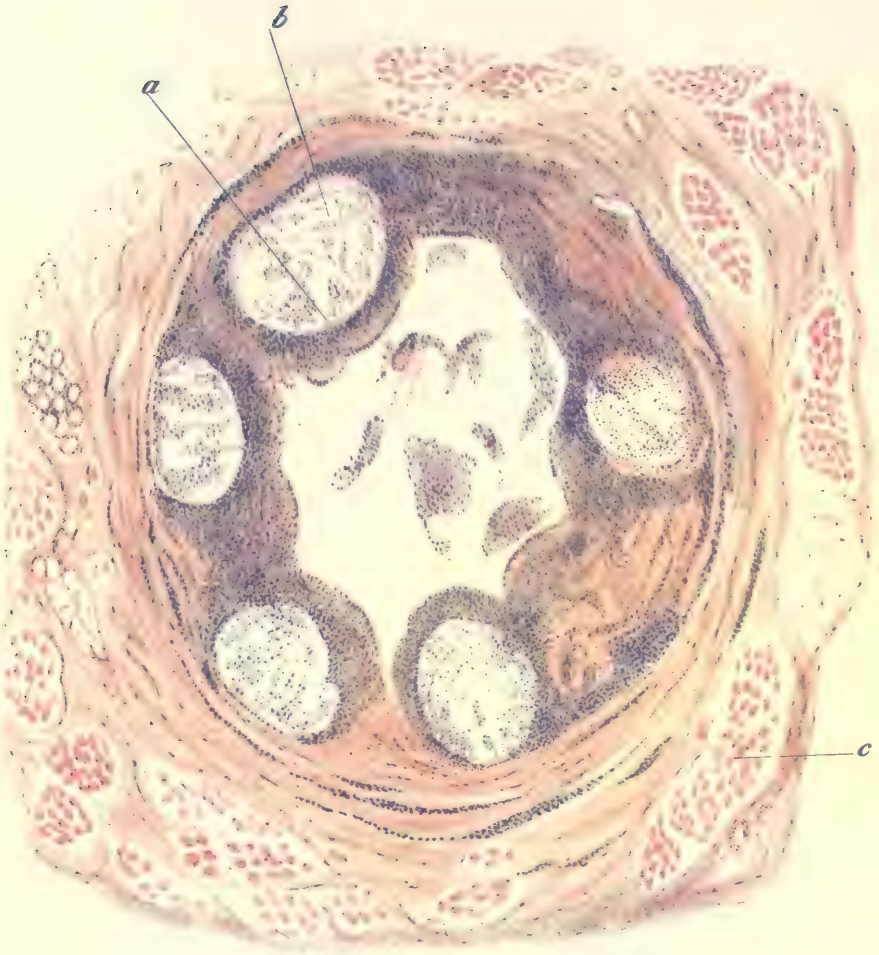
Thyreoglossal Cysts.—Cysts developing from remains of the thyreoglossal duct never become as large as branchial cysts. They lie in the median line of the neck in front of the hyoid bone, between it and the larynx, or below the larynx in the region of the jugulum. They form round, sharply defined, fluctuating tumors, which may be easily displaced, and are covered by normal skin. Although they are freely movable, it can always be noted upon palpation that they are attached to the deeper tissues, and often the cordlike attachment can be traced to the hyoid bone. This cord, which may so often be palpated, is the remains of the thyreoglossal duct. If the duct remains patent and ruptures upon the surface of the neck, a median cervical fistula forms.

The lining of the cysts and fistulæ developing from remains of the thyreoglossal duct differ. If they develop from the lower part of the duct they will be lined with ciliated epithelium; if from the upper part (ductus lingualis), with mucous membrane like that of the mouth. The walls of the cysts may contain mucous follicles.

Some of the difficulties in making a diagnosis of branchial cysts have already been mentioned in discussing dermoids. The large, distinctly fluctuating cysts may resemble congenital, cystic lymphangiomas so closely that a definite diagnosis can be made only by microscopic examination. In median cysts the cord, which may often be palpated and extends to the hyoid bone, is of great diagnostic significance. Dermoid cysts are superficial and freely movable. Tuberculous abscesses are usually associated with enlargement of the neighboring lymph nodes. Branchial cysts may be most easily confused with small, non-lobulated lipomas occurring in front of the hyoid bone.

The ductus lingualis, a part of the thyreoglossal duct extending from the foramen cæcum to the hyoid bone, has a genetic relationship to small cysts occurring about the foramen cæcum and cysts, known as ranula, which develop in the floor of the mouth. Some ranulæ are lined with ciliated epithelium and develop from Bochdalek's tubules, which are evaginations of the ductus lingualis.

Cysts of the Urachus.—Cysts of the urachus, which are not frequent, develop from the embryonal canal connecting the bladder with the allantois. The urachus usually undergoes complete involution and be-



LATERAL BRONCHIAL FISTULA.

(*a*) Remnants of ciliated epithelium.
(*b*) Lymph follicle.
(*c*) Longitudinal musculature of the fistula.

comes closed to form the superior true ligament of the bladder. If the canal remains open, a urachal fistula is formed, from which urine is discharged. If the canal becomes only partially closed, small cysts, the size of a bean, or a very large cyst which contains yellowish fluid, develops.

Cysts of the Vitelline Duct.—Cysts may also develop from the vitelline duct, which up to the eighth week of embryonal life extends between the intestines and the yolk sac. Vitelline cysts, like cysts of the urachus, are rare. If the duct remains patent, a congenital fistula, from which mucus and intestinal contents are discharged, persists after the cord separates. If the umbilical end of the duct closes and the intestinal end remains open, a blind sac (Meckel's diverticulum), which empties into the ileum, persists. If the intestinal end closes, and the umbilical end, from which the mucous membrane protrudes, remains open, an umbilico-vitelline diverticulum is formed. If both extremities become closed, but the intermediate part of the duct remains patent, a vitelline cyst (enterocystoma) forms as the secretion is poured out. The walls of the cysts, like those of the fistulæ and diverticula, are composed of regularly and irregularly arranged bundles of smooth muscle fibers. They are lined with epithelium which corresponds histologically to either the adult or embryonal types of intestinal epithelium. These cysts are found within the abdominal wall at the level of the umbilicus, and in front of the peritoneum, or within the abdominal cavity. When they occur within the abdominal cavity they are sometimes adherent to the parietal peritoneum; at other times to intestinal loops or are situated within the mesentery. The occasional occurrence of multiple cystomas suggests that some of these tumors may develop from displaced portions of germinal tissue, which later forms the intestines (Borst).

In the treatment of congenital epithelial cysts the cyst wall should be completely enucleated. This may be difficult, as the cyst may have contracted adhesions with the surrounding tissues at different points.

LITERATURE.—*Hildebrand.* 1. Ueber angeborene epitheliale Zysten und Fisteln des Halses. 2. Ueber angeborene zystische Geschwülste der Steissgegend. Arch. f. klin. Chir., Bd. 49, 1895, p. 167.—*Fr. König.* Ueber Fistula colli congenita. Arch. f. klin. Chir., Bd. 51, 1896, p. 578.—*Lexer.* Ueber die Behandlung der Urachusfistel. Arch. f. klin. Chir., Bd. 57, 1898, p. 73;—Magenschleimhaut im persistierenden Dottergang. Arch. f. klin. Chir., Bd. 59, 1899, p. 859.—*Nasse.* Ein Fall von Enterokystom. Arch. f. klin. Chir., Bd. 45, 1893, p. 700.—*v. Recklinghausen.* Urachuszyste. Deutsche med. Wochenschr., 1902, No. 34, Vereinsbeilage, p. 266.—*Roth.* Ueber Missbildungen im Bereich des Ductus omphalo-mesentericus. Virchows Arch., Bd. 86, 1881, p. 371.—*Sultan.* Zur Kenntnis der Halszysten und -fisteln. Deutsche Zeitschr. f. Chir., Bd. 48, 1898, p. 113.

CHAPTER II

CARCINOMAS

MALIGNANT epithelial new growths are grouped under the term carcinoma or *cancer*. They are characterized by an infiltrating growth, destroying the tissues which are invaded.

Relation Between Parenchyma and Stroma.—The relation between the parenchyma and stroma varies in different tumors and in different fields of the same tumor. The epithelial cells composing the parenchyma differ morphologically and in their arrangement, depending upon whether they have developed from the skin, mucous membranes, glandular viscera, embryonal epithelial rests, from preëxisting fibroepithelial tumors, or from epithelial cysts. In spite of the rapid multiplication of the cells in a carcinoma, they preserve the characteristics of the parent cells; for example, the cells of a carcinoma arising in skin become cornified; the cells composing carcinomas arising in the gastrointestinal tract, liver, and thyroid gland secrete mucus, bile, and colloid respectively. The stroma, or connective-tissue framework, consists of netlike trabeculæ varying in thickness and firmness. The stroma consists partly of newly formed connective tissue, partly of the tissues which have been invaded. It may therefore contain, besides old connective tissue, muscle, the parenchyma of the viscera involved, bone, etc.

Scirrhus and Medullary Forms.—If the stroma predominates the carcinoma is hard and resistant and tends to undergo cicatricial contraction. If the parenchyma predominates over the stroma the tumor is soft. The first form, which is called *scirrhus*, never becomes as large as the latter, which is called a *medullary carcinoma*. The intermediate form is usually called the *carcinoma simplex*.

Clinical Appearance.—A carcinoma may appear in a number of different forms, which depend more upon the tissues in which the carcinoma originates than upon the arrangement of the cells composing it; for example, carcinomas developing within the viscera are usually nodular, while those developing in the skin or mucous membranes tend to form tuberculated, cauliflowerlike, papillary, or polypoid growths, associated with a flat infiltration of the surrounding tissues and the formation of deep, craterlike ulcers.

Histogenesis.—There is no single conception among authorities concerning the histogenesis of carcinoma. After Virchow's teaching concerning the connective-tissue origin of carcinoma had been disproven by the brilliant researches of Thiersch, Waldeyer, Hauser, and others, Köster demonstrated that some of the tumors regarded as carcinomas

developed from the endothelium of blood vessels, and were, therefore, really of a connective-tissue nature. For this reason these tumors have been separated from carcinomas, sometimes being classified with sarcomas, at other times being regarded as a separate group (*endotheliomas*). Ribbert has ascribed to the connective tissue a very significant rôle in the development of carcinomas. According to him, the proliferation of the connective tissues is the cause, which leads to the irregular, atypical proliferation of the epithelial cells and the invasion of the tissues.

Squamous-cell Carcinoma.—It is the generally accepted view that in a squamous-cell carcinoma the proliferation begins in the germinal layer of the epidermis. At the point at which the carcinoma develops the cells multiply rapidly, preserving their embryonal characteristics and possessing irregular—the so-called pathological—karyokinetic figures, which may be easily explained upon the basis of excessive growth. The proliferating cells extend in all directions, raising and casting off the normal cells and sending down conelike processes into the deeper tissues and toward all sides, piercing the basement membrane, which normally separates the epithelial cells from the underlying structures. By the continued budding of these conelike processes, which are usually solid, a number of new processes are sent out into the tissues, so that eventually the point at which the carcinoma develops and its numerous processes resemble the roots of a plant. Naturally in microscopical sections the columns of cells which have been cut transversely or obliquely appear as separate islands of epithelium or as alveoli. True alveolar formation occurs if a group of cells becomes constricted off from the epithelial process; but this is rare, as usually the groups of cells which appear as separate would be found to be connected with the large epithelial downgrowths if serial sections were made.

Hauser and Petersen have succeeded in demonstrating clearly the method of growth by means of reconstruction in wax, using Born's method, which has been employed so extensively in reconstructing embryos. They have shown by this method that growth begins simultaneously at a number of points closely adjacent to each other.

According to the opinions of many authorities the cells of glands—for example, in carcinomas of the skin—the cells of the hair follicles, and of the sebaceous glands, and perhaps even of the sweat glands are involved in the growth (Borst). According to Ribbert, these observations are incorrect, the histological picture having been wrongly interpreted, as the epithelial cells surround the glands and appear to develop from them, while in reality they do not.

Changes in Connective Tissue.—While the proliferating epithelial cells are invading the cutis and subcutaneous tissues, and are produc-

ing a pressure atrophy of the normal tissues, the connective tissues do not remain inactive. Proliferative changes occur in the connective tissues, similar to those in mild inflammation, which lead to the formation of a fibrillar connective tissue rich in blood vessels. Atrophy and proliferation occur about the invading epithelial processes, which become surrounded with remains of the tissues that have been invaded and by those newly formed. When a carcinoma invades the skin, mucous membrane, or viscus, the preëxisting stroma forms part of the stroma of the tumor, while the stroma of the part of the tumor which develops above the surface of the skin, mucous membrane, or viscus is always newly formed.

A reactive proliferation occurs in all tissues invaded by carcinoma (especially in bone), as is demonstrated by the study of metastases.

According to Hauser, the growth relations in carcinomas composed of cylindrical cells, which may develop from any mucous membrane or embryonal rest composed of cylindrical cells, are the same as those described above in squamous-cell carcinoma. In cylindrical-cell carcinoma the epithelial processes are not solid, but are provided with a lumen and resemble, histologically, a gland.

Carcinomas developing from glandular epithelium grow in much the same way as described in the preceding paragraph. Sometimes the epithelial downgrowths have a lumen, at other times they do not. The normal glands are surrounded and compressed by the carcinomatous tissue, which spreads out in all directions.

The view generally accepted at the present time concerning the development of carcinoma is especially combated by Ribbert. According to him, carcinomas develop because of the weakness of the tissues in proximity to the cells, which are then no longer able to offer resistance to the downgrowth of proliferating epithelium, as they normally do. As a result of this weakness of the subepithelial connective tissues the cells break through the basement membrane and invade the surrounding tissues.

Borst admits that not infrequently groups of epithelial cells become separated or displaced as a result of the inflammatory proliferation of connective tissues, and that the epithelium may develop, but in his opinion the initiative to the proliferative processes lies in the epithelium. This is the opinion of the majority of pathologists at the present time.

Mode of Growth.—There is considerable difference of opinion among authorities as to the way in which carcinomas grow. Ribbert, Borst, and others believe that a carcinoma begins in small, limited, epithelial area, and that the cells of this area proliferate and invade the surrounding normal epithelial and glandular tissue, which are thus destroyed, while Hauser, Beneke, and others think that the normal cells bounding the

area in which the carcinoma develops become transformed into carcinoma cells and that the tumor enlarges by peripheral apposition. This theory of the transformation of normal epithelium into carcinomatous tissue is based upon the microscopic changes in the surface and glandular epithelium found at the margin of the carcinoma. It is supposed that there is an irritation originating at the point primarily involved which effects this transformation of normal into carcinoma cells. According to Ribbert's investigation, the proliferation occurring in the epithelial cells at the margin of a carcinoma is to be regarded as a reactive inflammation similar to that occurring in connective tissues which have been invaded by carcinoma cells, and not as a transformation of normal epithelial cells into carcinoma cells.

The same type of epithelial proliferation, even atypical, may occasionally be demonstrated in different chronic inflammatory processes. This type is found, especially in tuberculosis of the skin (Friedlaender), without indicating a carcinomatous degeneration, or changes which lead to carcinoma formation. It should also be remembered that groups of carcinoma cells may invade the surface epithelium and surround the normal gland tubules, so that normal glandular alveoli may be confused with the newly formed alveoli found in carcinomas.

Infiltrating Growth.—Carcinomas have almost exclusively an infiltrating growth. Carcinomas of the skin and mucous membranes extend along the surface and invade the deep tissues, and may proliferate to form growths which extend above the surface of the skin or mucous membrane. If the carcinoma develops in a viscus, the new growth extends in all directions. The carcinoma cells, as the tumor enlarges, displace the tissues and invade the delicate lymphatic space and vessels. In this way the door is opened for the formation of early and extensive metastases.

Lymphatic Metastases.—The carcinoma cells, after having invaded the lymphatics, may be detached by the lymph stream and carried into the tissues surrounding the primary focus, or they may invade the latter by means of their own amœboid movements. When the cells invade the surrounding tissues, small, red, firm nodules which never become larger than a pea develop in a wide area surrounding the primary focus. The development of these small nodules in the skin is often associated with carcinoma of the breast—the so-called lenticular carcinoma. This process is called the *local formation* of metastases or the dissemination of the carcinoma into the surrounding tissue. The growth extends in all directions along the lymphatic vessels, and the cutis surrounding a new growth may become almost symmetrically infiltrated. Often the boundaries of such an infiltrated area are sharply defined and scalloped and the area is reddened. The clinical picture then resembles very

closely that of erysipelas. [Occasionally in carcinoma of the skin or breast, reddened, indurated lines are seen running out from the primary focus. If the primary lesion is ulcerated it is frequently difficult to decide whether the lymphangitis is of a carcinomatous or bacterial origin. If of bacterial origin, it will usually subside after the application of an alcohol compress or moist dressing. It is important to determine the nature of the process as the indication concerning the extent of the proposed operation, and the prognosis depend upon the nature of the lymphangitis.]

Frequently the carcinoma cells are carried into the adjacent lymph nodes without becoming attached to the walls of the lymphatic vessels. [It should be remembered in this connection that any trauma of the endothelium of the lymphatics favors the attachment of carcinoma cells in the lymphatic vessels and the development of nodules. Extensive formation of secondary nodules in the lymphatic vessels is frequently observed in carcinoma of the breast after an osteopath or masseur has attempted to massage away the tumor.]

In the majority of cases of carcinoma metastases develop early in the regional lymph nodes. The cells are first carried into the cortical sinus, then into the follicles, and are finally deposited in the medullary portion of the nodes. The nodes soon become filled with the rapidly multiplying carcinoma cells, and the lymphoid tissue is compressed and destroyed. The lymph nodes when involved become indurated and enlarged. The induration is quite characteristic and is of great diagnostic significance. Upon section of small lymph nodes but recently involved, yellow or white foci which fuse with each other may be seen within a grayish red tissue. When the lymph nodes have been involved for some time they become transformed into carcinomatous masses.

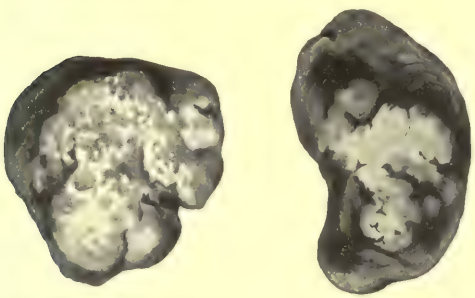


FIG. 410. — METASTATIC FOCI IN THE AXILLARY LYMPH NODES SECONDARY TO A CARCINOMA OF THE BREAST.

The carcinoma cells later break through the capsule of the lymph nodes, and adjacent nodes become fused to form large nodular tumors. The surrounding loose connective tissues then become invaded. If the skin covering a carcinoma becomes infiltrated, it finally ulcerates, and a nodular mass, the center of which degenerates to form a craterlike, foul-smelling ulcer, projects above the surface of the skin. The extension of the carcinoma cells along the perivascular lymphatics of large ves-

sels is dangerous, as severe, usually fatal, hæmorrhage follows the invasion of the different coats of the arteries and thrombosis and dissemination of the growth through the blood stream follow the invasion of the walls of the vein.

Involvement of lymph nodes which ordinarily do not receive lymph from the area involved, but are out of the path usually traveled by the lymph, has been explained by retrograde embolism.

It is more probable in these cases that the carcinoma cells proliferate along the lumina of the vessels, forming a continuous mass, than that the lymph stream becomes reversed as the result of the occlusion of one of the larger lymphatics and that the cells are carried to the nodes by the lymph. As the result of this carcinomatous proliferation along the lumina of the lymphatic vessels, white cords are formed in carcinoma of the peritoneum which may frequently be seen extending from one diseased lymph node to another beneath the membrane. The white cords correspond to lymphatic vessels filled with carcinoma cells. White, netlike cords may also be seen beneath the visceral pleura, associated with metastatic foci in the lymph nodes or lungs.

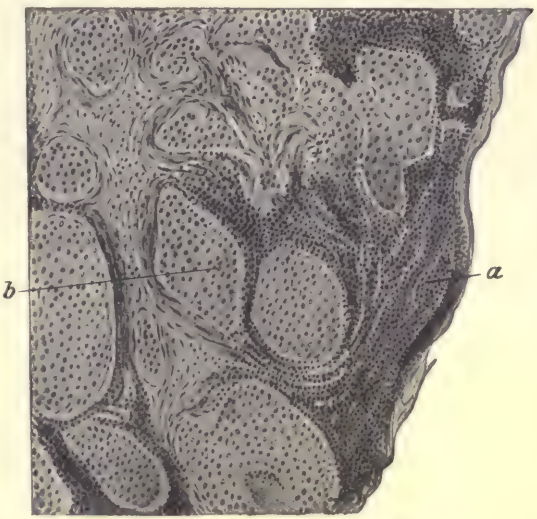


FIG. 411.—CARCINOMATOUS LYMPH NODES. *a*, Remains of lymphoid tissue; *b*, groups of carcinoma cells.

Hæmatogenous Metastases.—Hæmatogenous metastases may follow involvement of the lymphatics, occurring when carcinoma cells invade the thoracic duct or are carried by the lymph stream into the veins.

Direct hæmatogenous metastases, occurring earlier than the secondary above described, are rare but more dangerous. They follow the invasion of the walls of veins, emboli of carcinoma cells being carried into the general circulation. Direct hæmatogenous metastases may develop from carcinomatous lymph nodes as well as from the primary focus.

The lungs and liver are the first filters of the systemic and portal circulations respectively. The one or the other, depending upon the position of the primary tumor, is first involved in hæmatogenous metas-

tases. If the carcinoma cells are carried from these organs, they are deposited in the different viscera, the skin, and bones.

It is worthy of note that all carcinomas do not have the same tendency to the formation of metastases in bone. This tendency is most pronounced in carcinomas of the breast, prostate and thyroid gland, and in hypernephromas. The metastases usually develop in the ends of long, hollow bones or in the bodies of the vertebra, which even in adults are

FIG. 412.—SCIRRHUS OF THE BREAST WITH SECONDARY NODULES IN THE SKIN.

richly supplied with blood vessels. The bone, at the point at which the metastasis develops, either undergoes an atrophy resulting in a spontaneous fracture, or, when the vertebræ are involved, in a gibbus, or it is stimulated to the formation of considerable new bone (osteoplastic carcinoma).

In carcinomas, metastases usually develop by way of the lymphatics, while in sarcomas metastases usually develop by way of the blood stream. This difference depends upon the peculiarities of growth of these two classes of tumors; perhaps also upon the fact that carcinoma cells may die in the blood stream.

Histology of Metastatic Growths.—The cells found in the metastases preserve the characteristics of those composing the primary growth. They may, however, lose their typical arrangement and their properties of forming keratin or mucus in the metastases, these changes sometimes being indicative of very rapid proliferation, at other times of degeneration.

Implantation Carcinoma.—Other peculiar methods of extension which occasionally occur must be attributed to the implantation or accidental transplantation of carcinoma cells. This so-called *implantation carcinoma* develops upon opposing surfaces of the lips, of the tongue and cheek, and of the labia, upon the inner surfaces of the thigh or the vocal cords. One surface is primarily involved, the opposing surface secondarily.

It has been attempted to explain the development of multiple carcinomas in the gastrointestinal tract, in the respiratory passages, and urinary tract by the transplantation of carcinoma cells carried from a primary tumor situated higher (e. g., carcinoma of the tongue and œsophagus, carcinoma of the œsophagus and stomach, of the pharynx, tongue and larynx, of the kidney, and urinary bladder). It is exceedingly difficult to determine whether these multiple carcinomas have developed as the result of the implantation of carcinoma cells from a primary tumor. In the first place multiple carcinomas which undoubtedly have developed absolutely independently of each other occur, and in the second place it is possible that the tumors may have developed by retrograde lymphogenous embolism. The latter can be positively determined only when a lymphatic vessel filled with carcinoma cells can be found extending between the primary and secondary tumors.

Macroscopic Appearance upon Section.—Macroscopically a carcinomatous mass is grayish red in color and somewhat translucent upon section. Its boundaries are rather sharply defined against the surrounding tissue, but the tumor cannot be enucleated, as it is firmly attached. If there are no areas of hæmorrhage, necrosis, or softening, the cut surface of such a tumor is homogeneous.

Upon section there are two very important characteristics: (1) If the stroma is well developed, yellowish white nests of epithelial cells may be seen with the naked eye. These form small, round foci which resemble when expressed by digital pressure comedolike masses or vermicelli; (2) a white, milky juice—the so-called cancer milk—exudes from the cut surface, especially of soft carcinomas, and may be easily scraped up with a knife.

The appearances of different carcinomas differ, depending upon a number of different factors. If the tumor contains a large number of blood vessels the tissues will have a dark appearance. Carcinomas of

the mucous membranes and viscera, in which there is a production of large amounts of mucus, have a gelatinous, glassy appearance. The differences in macroscopic appearance also depend upon the character and extent of the regressive changes. Atrophy and necrosis are the most important regressive changes occurring in carcinomas. These are due in part to the imperfect nutrition of the rapidly growing, decaying tissues; in part to the obliteration of large vessels by the pressure of the tumor masses and occlusion of the vessels by carcinomatous thrombi.

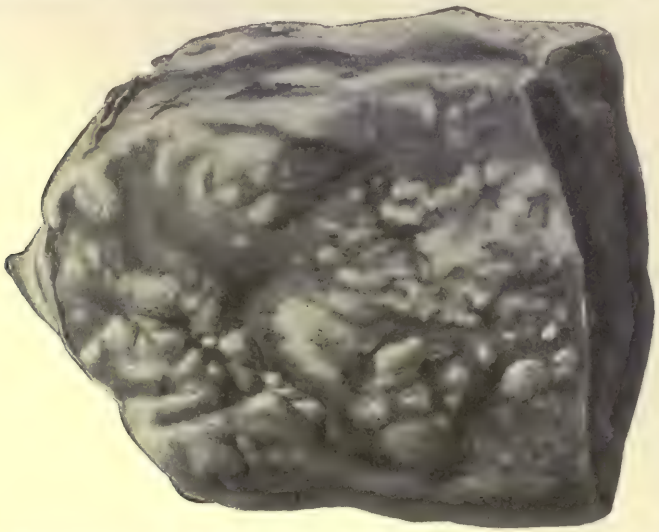


Fig. 413.—Metastatic Foci in the Liver, Secondary to a Carcinoma of the Rectum.

Degeneration of the cells is followed by softening. After the absorption of the liquefied masses, the carcinomatous area in a viscus becomes depressed and contracted forming the so-called "carcinoma navel." Degeneration occurring in the skin and mucous membranes, which have become infiltrated, leads to the formation of carcinomatous ulcers. Softening, liquefaction, and hæmorrhage lead to the formation of cysts, which should, however, be differentiated from the cavities occurring in cysts which have become carcinomatous. The tumor masses also may become calcified.

Age, Sex, and Frequency with which Different Tissues are Involved.—Carcinomas develop most frequently in middle or advanced life, the disease being most common in the fifth and sixth decennia. Not infrequently the disease occurs at an earlier age during the second and third decennia. The disease is very rare in children, but occasionally congenital carcinomas are observed, developing most frequently in teratoid tumors.

Carcinoma is more frequent in the female than male sex, the relative proportion being six to four. The great frequency of carcinoma in the female is due to the frequent development of these tumors in the female breast and in the organs of generation (Borst).

The statistics of different authorities concerning the frequency with which the different tissues and viscera are involved differ. According to Borst they are involved in the following order of frequency: skin, stomach, intestines (rectum), uterus, mammary gland, œsophagus, ovary, gall bladder, pancreas, lung, urinary bladder, larynx, liver, thyroid gland, tongue, kidney, and prostate. As a rule, but a single primary carcinoma develops.

The simultaneous development of multiple carcinomas, which are absolutely independent of each other, is the exception. Sometimes multiple independent new growths develop upon the skin or mucous membrane which is already involved by some other lesion—e. g., upon a leukoplakia, a senile seborrhea, an X-ray dermatitis, a xeroderma pigmentosum, a tuberculosis of the skin, a chronic eczema in both breasts following a chronic mastitis, in the intestinal mucous membrane, the seat of multiple polypi (polyposis). The multiple carcinomas in these cases have the same structural relations as the tissues from which they develop —for example, a carcinoma of the intestines composed of cylindrical epithelium may develop simultaneously with a carcinoma of the skin which is composed of squamous epithelium.

From a clinical viewpoint it is of advantage to classify carcinomas according to the tissues from which they develop—for example, into carcinomas of the skin, mucous membranes, and glands.

(a) CARCINOMAS OF THE SKIN

The majority of these carcinomas develop from the surface epithelium, and are, therefore, composed of squamous cells. More rarely they develop from the glands of the skin, being then composed of cylindrical cells.

Origin and Histological Characteristics.—As a squamous-cell carcinoma grows, solid processes composed of epithelial cells invade the subjacent tissues. These processes are composed of flat epithelium with oval nuclei. The origin of these cells is frequently indicated by the formation of protoplasmic bridges and cornification which give to these tumors typical histological pictures. In many carcinomas, especially in the superficial forms developing in the skin of the face, these characteristics are lacking (*vide* below). The young cubical or cylindrical cells corresponding to the germinal layers of the epidermis occupy the periphery of the downgrowths, while the older, flat, cornified cells are

found in the center. The cells are concentrically arranged, and in this way the *epithelial pearls*, so characteristic of squamous-cell carcinoma, are formed. If some of the cytoplasm of the cells becomes cornified while the remainder undergoes hyaline or fatty changes, peculiar bodies are formed within the cells which have often been regarded as parasites (Borst). Multinucleated giant cells may also be found in squamous-cell carcinomas. These cells which develop from the connective tissue lie between the stroma and the epithelium (*vide* Petersen).

Carcinomas developing from sebaceous glands are characterized by broad, clubbed downgrowths composed of flat epithelium which often has a glandular arrangement and is undergoing fatty metamorphosis.

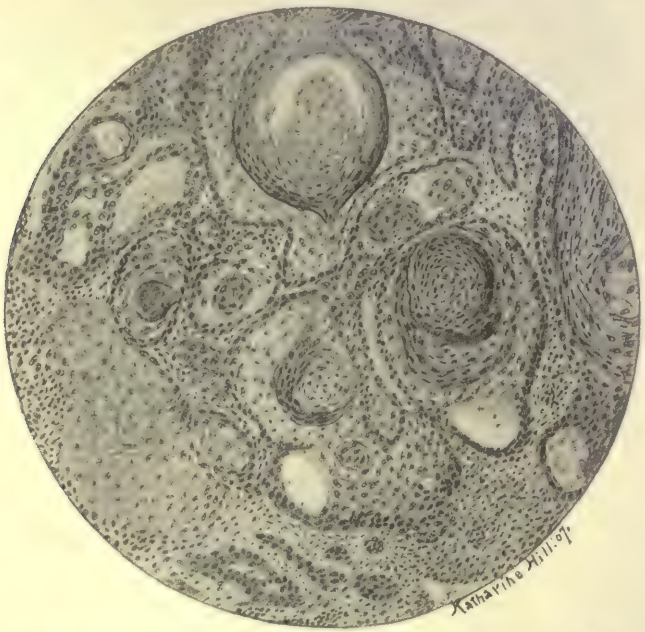


FIG. 414.—SECTION FROM AN EPITHELIOMA OF THE LOWER LIP, SHOWING EPITHELIAL PEARLS. (From Professor Bevan's Surgical Clinic.)

Carcinomas also occur in the skin, especially upon the face, which correspond in their clinical course to the squamous-cell carcinomas, occasionally even forming nodular tumors, but which are morphologically different. The columns of cells in these carcinomas are narrow and pointed, often containing a lumen. The cells are elongated and small.

Carcinoma Basocellulare.—The delicate, narrow columns of cells, corresponding in their distribution to the lymphatic vessels, give to these tumors a peculiar histological picture. Usually the cells do not become

cornified, and if cornification does occur it is limited to a few cells. The glandlike arrangement of the epithelial downgrowths is frequently so striking that Krompecher has spoken of these tumors as GLANDLIKE CARCINOMAS ARISING FROM THE SUPERFICIAL EPITHELIUM. Whether his view that these carcinomas develop from the basal cells, while the squamous-cell carcinomas develop from the prickle cells is correct or not, cannot be answered positively at present. There are a number of authorities who do not agree with him in this assertion. According to von Hansemann and Ribbert the division of carcinomas of the skin into basal- and prickle-cell carcinomas cannot be made. Ribbert regards the tumors under discussion as carcinomas of the skin, the cells of which do not become cornified; Borst regards them as endotheliomas. Borrmann groups the carcinomas of the skin, the cells of which do not become cornified, as CARCINOMAS OF THE CORIUM, for they begin to develop, as he has demonstrated, in the corium. He believes that they develop from multiple, misplaced groups of epithelial cells, or from displaced anlage of hair follicles, sweat and sebaceous glands.

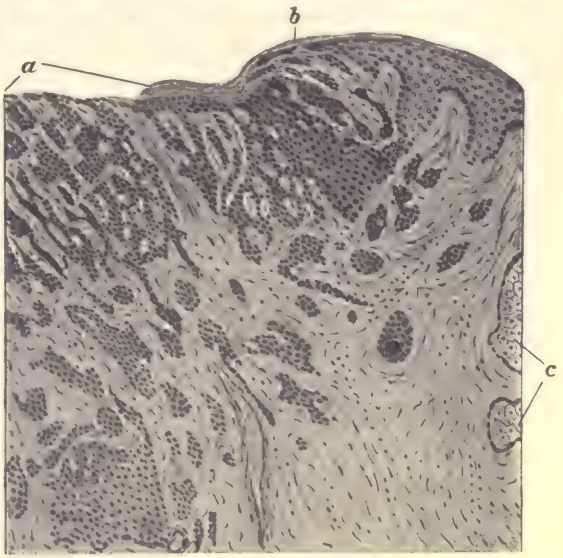


FIG. 415.—SUPERFICIAL CARCINOMA OF THE SKIN OF THE NOSE, THE CELLS OF WHICH DO NOT BECOME CORNIFIED. (Basal cell carcinoma of Krompecher, corium carcinoma of Borrmann.) *a*, Superficial ulcer; *b*, edge of the ulcer; *c*, sebaceous glands.

Most Common Sites for Development.—Carcinomas of the skin occur most frequently upon the face, more rarely upon other parts of the body, of which the scalp, the skin of the back of the neck, of the concha, the external auditory meatus, the external genitalia, the extremities, and the navel should be mentioned.

Predisposing Factors.—These tumors develop either from perfectly normal skin or from skin which has previously been the seat of some other lesion. Carcinomas may develop from benign fibroepithelial growths, such as warts, papillomas, cutaneous horns, hypertrophies of hair follicles, from adenomas of the sebaceous and sweat glands, from atheromas and dermoid cysts. Irritation associated with chronic inflammatory processes favors the development of these tumors. The following

well-known examples may be cited: Carcinomas developing after repeated attacks of erysipelas, from a chronic eczema of the scrotum or extremities produced by soot (chimney-sweep's carcinoma), from irritation of the skin of the hands and forearms occurring in workers in paraffin (paraffin carcinoma) and tar, and from syphilitic, tuberculous, varicose, and trophoneurotic ulcers. A carcinoma of the skin sometimes develops early in life upon a xeroderma pigmentosum—a congenital atrophy of the skin first described by Kaposi, which is associated with a peculiar distribution of pigment. Carcinomas of the skin develop very frequently in advanced life from a senile seborrhœa, which is most common upon the temporal and frontal regions, the cheeks, and the dorsum of the nose. The relationship between senile seborrhœa and the development of carcinoma has been noted by von Volkmann, Schuchardt, and von Bergmann especially.

Senile Seborrhœa.—Senile seborrhœa, which is usually the result of uncleanliness, is characterized by a proliferation of the epidermis, associated with a cornification of the cells and an accumulation of the secretion of the sebaceous glands between different layers of the proliferating epidermis, producing an occlusion of the lumina of the glands. The lesions appear as multiple, dark-yellow or brown scalelike deposits, which feel oily and are usually well circumscribed, or occasionally as wartlike growths. The scale may be removed if the surface is rubbed, the corium, which bleeds slightly, then being exposed. After the seborrhœic crusts have been removed a number of times, a superficial ulcer develops. If kept clean and treated with ointments, such an ulcer heals rapidly; if, however, it is neglected or scratched, it remains open. The ulcer associated with senile seborrhœa differs from that of a carcinoma in that it does not enlarge, remains superficial, and its edges do not become indurated.

Scars resulting from injuries and ulcers of the skin (syphilitic, tuberculous, varicose, neuropathic, burns), and chronic fistulæ may also afford the conditions which favor the development of carcinoma. Sometimes a carcinoma follows soon after an injury, developing in a recent scar. In these cases a single trauma must be regarded as the essential, or at least the exciting cause of tumor formation. A carcinoma may follow repeated injuries of the lip during shaving, developing usually at the mucocutaneous border of the lower lip. Fistulæ from which carcinomas occasionally develop usually have persisted for a number of years. These growths may develop from fistulæ about the rectum, from those associated with suppurating or tuberculous foci in bone, or remaining after the drainage of an empyema.

Carcinomas of the skin appear in three forms: the *superficial, deep,* and *papillary.*

(1) SUPERFICIAL CARCINOMAS OF THE SKIN

Appearance and Clinical Course.—These develop as small, firm, red nodules which are usually not observed at first, as they cause no pain. The attention of the patient is first attracted to the growth after it has attained considerable size or when the epithelium is cast off and the surface becomes covered with an unsightly crust. When the latter is removed repeatedly a gradual enlargement of the underlying ulcer may be noted. A benign, superficial defect in the skin, developing after an injury or the removal of seborrhœic crusts, may imperceptibly develop into carcinoma.

If the epithelium is retained for some time (this occurs especially in basal-cell or corium carcinomas), a superficial, platelike nodule forms in which frequently small cysts with clear contents may be recognized. The cysts are produced by a retention of secretion in, and subsequent dilatation of the glandlike downgrowths of cells found in basal-cell carcinomas, and those developing from sweat and sebaceous glands.

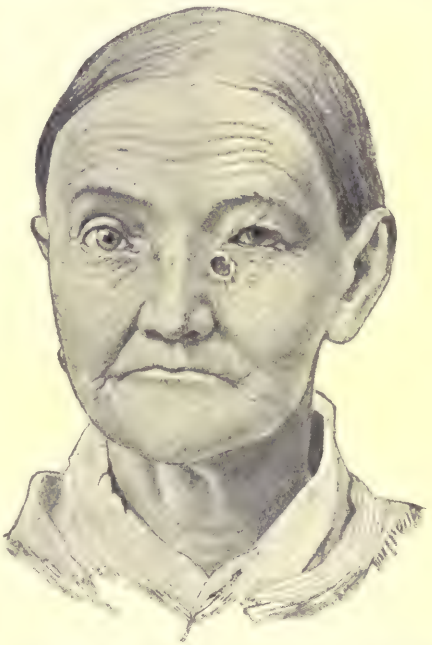


Fig. 416.—Superficial Carcinoma of the Skin.

The form and clinical course of superficial carcinomas are characteristic as long as the tumor remains superficial and extends within the cutis.

The borders of the ulcer are but little indurated, and the ulcer can be moved with the skin so long as the cells do not invade the deeper tissues. Invasion of the deeper tissue, which is usually accompanied by considerable pain, occurs only after the lesion has persisted a number of years. When the crust, consisting of dried secretion and carcinoma cells, is removed, the stroma, which carries the blood vessels and is easily injured, bleeds. The floor of the ulcer is red and but slightly fissured, as it does not extend deeply into the subjacent tissues.

As the growth invades the surrounding tissues a firm, wall-like, but narrow, border is formed which is covered by the raised, undermined epithelium, and is rather sharply defined against the floor of the ulcer.

As ulceration occurs the borders of the ulcer acquire a serpiginous or jagged outline and frequently appear undermined, as the carcinoma cells undergo more rapid regressive changes than the epithelium of the border of the ulcer.

If the carcinoma cells in the center of the ulcer degenerate completely, the proliferation of the stroma, followed by cicatricial contraction, becomes marked and radiating folds are formed which extend into the normal skin (Fig. 416), causing distortion of the eyelids and lips. [" These superficial carcinomas, frequently called ' rodent ulcers ' by American and English surgeons, may heal over at certain periods of the year. An old man presents himself in a clinic and states that an ulcer upon the face, undoubtedly of carcinomatous nature, becomes raw in cold weather and heals at certain periods of the year, being covered by a delicate bluish epithelium. The raised border surrounding such a scar still indicates the nature of the lesion. These ulcers may heal spontaneously, but the temporary healing is often attributed to some ointment which may have been applied shortly before the spontaneous but temporary healing occurred.

FIG. 417.—SUPERFICIAL CARCINOMA OF THE NOSE OF TEN YEARS' STANDING.

" The spontaneous healing is only temporary. Another ulcer soon forms which extends more quickly, and then it may be seen that the carcinoma cells were invading deeper tissues, even when the ulcer was apparently healing. No structures seem to resist the ravages of the disease, and most museums contain evidences of the hideous results of rodent ulcer upon the face, destroying the contents of the orbit and the bones of the nose, and laying bare the nasopharynx. Bands of fibrous tissue long resist the ulceration, and, although the vessels may be dissected out they are seldom if ever laid open. All this time the general health is not affected, there is little or no pain, unless the eyeball or

nerve trunks are involved, and the lymphatic nodes remain quite uncomplicated.''—Allbutt's '' System of Medicine,'' Vol. IX, p. 843.]

Lupuslike Carcinoma.—Von Bergmann has described a peculiar form of superficial carcinoma occurring in the skin of the temporal regions which he has described as lupuslike carcinoma. It begins with the formation of small nodules in the skin. These disappear without ulcerating, leaving a scar uncovered by hair. New nodules later develop about the scar, finally encircling the latter. After the disappearance of these nodules with resulting scar formation, new nodules develop about the periphery, so that finally a large area of skin is involved.

These superficial carcinomas, which occur most frequently upon the face and scalp (cheeks, eyelids, nose, temporal regions, forehead, or external ear), may persist for a number of years before they extend deeper. Eventually such a growth may transform the deeper tissues into a friable, ulcerating mass, and rupture into the cavities of the face or destroy the bones of the skull and expose the dura mater.

König describes a superficial carcinoma occurring in a woman ninety years of age, which involved only one half of the face after persisting for twenty-five years, and another case (represented in Fig. 417) occurring in a man which pursued a clinical course of ten years' duration before it produced a complete destruction of the nose.

Fig. 418.—Superficial Basal Cell Carcinoma of the Skin (Companion to Fig. 415). Regarded for some time as a syphilitic, later as a tuberculous, lesion.

A superficial carcinoma of the skin grows very slowly, usually in the form of a superficial ulcer with slightly elevated, serpiginous, or undermined borders, with a slightly fissured, uneven base which bleeds easily when the crust covering it is removed. Such a carcinoma has a great tendency to cicatricial contraction, scar formation, and apparent healing. If neglected a superficial carcinoma finally involves the deeper tissues.

Metastases.—The adjacent lymph nodes are involved late, usually not before the lesion has involved the deeper tissues. When the lymph nodes are involved they become enlarged and indurated.

Hæmatogenous metastases occur in neglected cases only.

Diagnosis.—The diagnosis of superficial carcinomas of the skin is usually easily made when the characteristics above mentioned are kept in mind. The chronicity of the ulcer, the absence of a dusky margin, and a serpiginous outline at once distinguish a rodent ulcer from the ulcer of tertiary syphilis. The resemblance between an ulcerated gumma and a superficial carcinoma may be very close, and it is often difficult to distinguish between the two without a microscopic examination. A rodent ulcer in a syphilitic subject may be curiously modified.

A tuberculous ulcer has flat, irregular borders which are often undermined for some distance, and a distinctly reddish or yellowish floor containing caseated masses and tubercles. Single, isolated syphilitic and tuberculous ulcers are the exception. They are usually associated with other lesions closely adjacent or upon other parts of the body, while single lesions are the rule in carcinomas. Superficial ulcers developing from adenomas of the sweat glands and following seborrhea never have indurated, thickened borders.

Treatment.—Thorough excision, carried into healthy tissues, is the only successful treatment, as rapid, safe, and permanent healing cannot be secured by any of the other procedures which have been recommended, such as cauterization with the hot iron, different caustic solutions and pastes, and the use of Röntgen and radium rays. If the excision is properly performed, the carcinoma recurs in a very few cases (in 4.5 per cent, according to Borrmann). Extensive and deep recurrences follow so frequently the apparent healing, which may also occur after the use of the dry aseptic dressing (von Bergmann), produced by the agents above mentioned, that a word of warning should be spoken. Lexer has seen a number of cases in which the carcinoma became inoperable after having been treated for a number of months with Röntgen and radium rays.

The defect resulting from the excision of the ulcer, which should be carried well into the healthy tissues, should be repaired by a plastic operation. Defects upon the forehead and body should be covered with epidermal strips. The surface of bone, when invaded, should be chiseled away. The eye should be enucleated as soon as a carcinoma developing upon the lid invades the orbit and the bulb.

Diseased or suspicious lymph nodes, together with the connective tissue and fat surrounding them, should be radically removed. Often the enlargement of the regional lymph node is of an inflammatory nature, being due to the absorption of infectious materials from the ulcer. Sometimes the nodes are tuberculous.

Carcinomas of the face may finally extend deeply, destroying the bone and exposing the dura mater or invading the ethmoid. They then

become inoperable. Caustic pastes (zinc chlorid), the use of the actual cautery and of compresses of hydrogen peroxid solution are indicated to retard the extension of the disease and overcome the odor associated with putrefaction.

(2) DEEP CARCINOMAS OF THE SKIN

Origin.—These carcinomas develop from small, round nodules which, when situated in the deeper tissues, originate in the glands of the skin (sebaceous glands, hair follicles, perhaps also sweat glands) or from superficial carcinomas which have existed for a long time. They have the characteristics of new growths, which are often almost completely wanting in the superficial carcinomas of the skin. They occur most frequently upon the face, involving the nose, the eyelids, and the mucocutaneous border of the lips. When the lips are involved, the clinical picture resembles very closely that of carcinoma of the mucous membranes. These tumors are rarely found on other parts of the body.

Appearance and Clinical Course.—They rapidly invade the surrounding tissues and degenerate to form ulcers, the bases of which are indurated and fused with the underlying tissues (fascia, bone, etc.). Fissures, spaces, and craterlike depressions, covered by crusts and degenerated epithelium, and in which easily bleeding carcinoma tissue may be found, are present in the floor of these ulcers. Plugs of carcinoma tissue may be expressed from the ulcer if lateral digital pressure is made. The edges of the ulcer are raised and definitely infiltrated. They are hard to the touch, and the induration may extend to the tissues beneath. The edges of the ulcer are pinkish in color and often marked by dilated capillaries. The discharge is serohæmorrhagic in character and filled with the decomposing products of cellular débris. If the ulcer is large, the secretion has a peculiar and offensive odor. Severe hæmorrhages may follow the ulceration of large arteries in the deeper parts of the new growth.

Nodular or wartlike projections may develop in the floor of such an ulcer later in the clinical course, and project above the level of the surrounding skin, forming a transitional stage to the papillary form of carcinoma of the skin.

Metastases.—The regional lymphatic nodes become extensively involved and transformed into large nodular tumors early in the course of the disease.

Hæmatogenous metastases are not rare.

Treatment.—The course of this form of carcinoma is so rapid that the diagnosis must be made early if the treatment is to be effectual. Any wart or scar occurring in old people, which begins to enlarge rapidly and ulcerate, or rapidly developing and ulcerating warts or papillomas, which form upon an inflammatory base (for example, the warts

developing upon the chronic eczema occurring in chimney sweeps), should arouse suspicion of malignancy, even when the definite characteristics of carcinoma are wanting.

Removal of the growth by excision is the only treatment that should be considered. The excision should be carried well into healthy tissue, and the regional lymph nodes should be removed. It is frequently necessary to resect the bone adjacent to the growth.

The same methods should be employed in the treatment of inoperable cases as have already been described in discussing superficial carcinomas of the skin.

(3) PAPILLARY CARCINOMAS OF THE SKIN

Appearance and Clinical Course.—These carcinomas are characterized by the early growth of the tumor tissue above the level of the skin. They usually develop from small nodules, wartlike growths, or from carcinomatous ulcers, and usually appear as firm pedunculated or sessile tumors with hard infiltrated bases, overhanging borders, and nodular, fungoid, cauliflowerlike or papillomatous surfaces in which deep depressions may be seen and which are frequently covered by horny masses or crusts composed of dried secretion. The skin usually stops abruptly at the margin of such a growth.

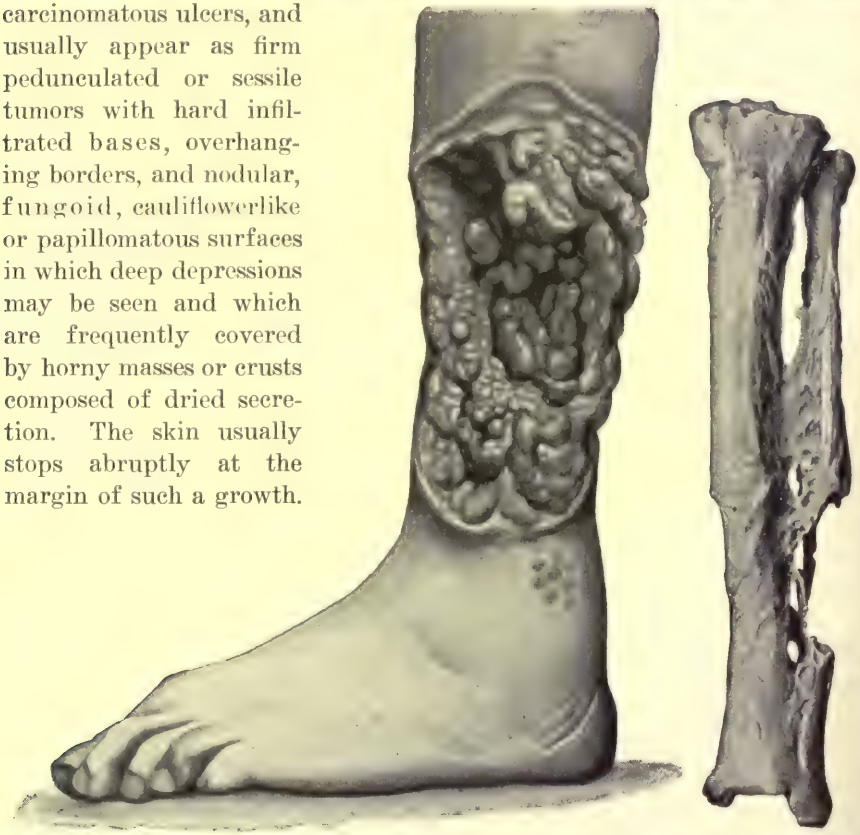


FIG. 419.—NODULAR CARCINOMA WHICH DEVELOPED UPON A VARICOSE ULCER OF THE LEG. The fibula has been partially destroyed by the growth.

Upon section, long branched papillary growths are seen developing from the carcinomatous masses, which in the beginning, at least, do not extend far into the subjacent tissues.

Carcinomas of the skin of the extremities (Fig. 420) and of the penis, and carcinomas developing in the mucous membrane of the glans and inner layer of the prepuce produce the same clinical

FIG. 420.—CAULIFLOWERLIKE CARCINOMA OF THE BACK OF THE HAND WHICH DEVELOPED IN A SCAR ONE AND A HALF YEARS AFTER AN INJURY. (Male patient sixty years of age.)

picture. Carcinomas of the skin of the face assume this picture more rarely.

This form of carcinoma develops most frequently from fibroepithelial growths and in scars and ulcers; occasionally from atheromas and dermoid cysts.

Rapid enlargement of a wartlike growth should arouse suspicion of malignancy. When a papillary carcinoma develops upon an old ulcer, small, hard nodules first appear within the flabby granulations, and then infiltration and induration of its edges quickly follow.

Metastases.—The adjacent lymph nodes rapidly become involved. General metastases may occur.

Treatment.—If early and extensive excision is performed, recovery without metastases may occur. In papillary carcinomas of the extremity which have invaded and passed through the fascia, amputation may afford the only hope of permanent cure. Excision carried well into healthy tissues should be attempted only when the growth is still limited and is not adherent to the underlying tissues (tendons, bone). The penis should always be amputated when a carcinoma involves this organ.

The other rules for the treatment are the same as described in discussing other forms of carcinoma of the skin.

(b) CARCINOMAS OF THE MUCOUS MEMBRANES

Clinical Forms.—Carcinomas of the mucous membranes appear in a number of different forms; sometimes developing as pedunculated or sessile, nodular tumors, sometimes as fungous, cauliflowerlike, papillomatous, or villous growths, sometimes as superficial or deep ulcers, and finally as diffuse infiltrations.

They are very different morphologically. The difference depending mostly upon whether the tumor develops from mucous membranes covered with squamous or cylindrical epithelium, or whether it develops from glands within the mucous membranes.

Squamous-cell Carcinomas.—Squamous-cell carcinomas with cornification, similar to those of the skin, occur most frequently upon the tongue and lips. The lower lip is more frequently involved than the upper lip. They also occur in all parts of the mouth cavity (cheeks, floor of the mouth, soft palate, tonsils), in the larynx, the œsophagus, the cardiac end of the stomach, in the vagina, the cervix of the uterus, upon the mucous portions of the labia, the prepuce, and the glans penis.

FIG. 421.—CARCINOMA OF THE NECK, SECONDARY TO A CARCINOMA OF THE LIP. Involvement of the skin following extension from lymph nodes. (From Professor Bevan's Surgical Clinic.)

Carcinomas, the cells of which become cornified, may also develop from the transitional epithelium of the urinary passages.

In the rare cases in which the squamous-cell carcinomas develop in mucous membrane covered with cylindrical epithelium (gall bladder, stomach, trachea), it must be supposed that they develop from dis-

placed embryonal tissue consisting of squamous cells or from epithelium which has undergone metaplasia as the result of chronic inflammation or long-continued pressure.

Predisposing Factors.—Often some other lesion of the mucous membrane prepares the way for the development of a carcinoma. Carcinomas of the mucous membranes, like carcinomas of the skin, may develop from fibro-epithelial growths, from ulcers of all kinds (especially the tuberculous and syphilitic), from scars and wounds. Injuries produced by the sharp points of carious teeth and the disease known as *leucoplakia* or *psoriasis linguæ* are of about equal importance as etiological factors in the development of carcinoma of the tongue and cheek.

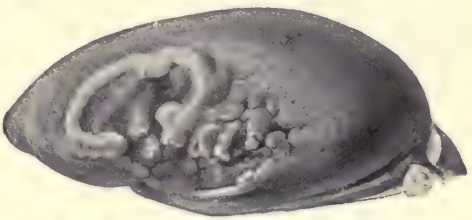


Fig. 422.—Carcinoma of the Tongue.

Leucoplakia.—Leucoplakia is characterized by the gradual, painless formation of white, light gray, or opal, slightly elevated plaques, which are observed most frequently upon the dorsum of the tongue, and then, in order of frequency, upon the margins and lower surface of the tongue, upon the mucous membrane of the cheek, especially near the angle of the mouth, upon the lips, and more rarely upon the soft palate. The plaques, which usually are sharply defined, are irregular in shape and vary in size. These epithelial plaques, which are, as a rule, firmly attached, are formed by the excessive proliferation and cornification of the epithelium. Not infrequently epithelial processes are found within them which extend through the basement membrane to penetrate the submucous tissue, indicating that a carcinoma has already commenced to develop.

Leucoplakia may develop in middle life and remain entirely harmless as long as thirty years, or longer. At any time, however, a nodular induration associated with pain may develop in the mucous membrane beneath the white plaque. The induration may be due to different causes. In the majority of cases the painful induration is due to a transitory inflammation of the mucous membrane beneath the white patch, which has become rough and fissured. Sometimes the nodule enlarges and becomes more indurated, instead of subsiding, indicating the beginning of carcinoma formation. In many cases the development of multiple carcinomas at different points in the tongue, cheeks, and lips may be observed after long intervals. Benign papillomas may develop upon a leucoplakia as well as carcinomas (von Bergmann) (Fig. 423). The development of nodules upon a leucoplakia should always

arouse suspicion. When a nodule develops it should be removed and examined microscopically in order to make a positive diagnosis, and the proper treatment should then be instituted. Benign growths should be removed, as they may become malignant at any time.

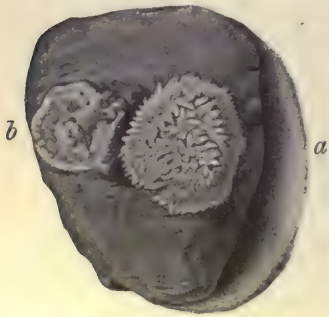


FIG. 423.—*a*, PAPILLOMA, CORNIFICATION OF THE CELLS COVERING THE GROWTH IS MARKED. *b*, CARCINOMATOUS ULCER WITH INDURATED, CRATERLIKE EDGES. The papilloma and carcinoma are separated by an area of leucoplakia. (Von Bergmann's Handbook of Practical Surgery.)

Leucoplakia occurs almost exclusively in men, most frequently in those who smoke to excess. It is rare in women, even in those addicted to smoking. Tobacco must therefore have the same relation to carcinoma of the mucous membranes that tar and paraffin have to carcinoma of the skin (von Bergmann).

The treatment of leucoplakia is successful only when smoking is stopped. It consists usually of the use of non-irritating mouth washes. When the plaque becomes fissured and an inflammatory zone develops about the lesion, the actual cautery is to be recommended. Hard nodules which continue to enlarge indicate the beginning of carcinoma formation. These should be excised, and the spindle-shaped or cuneiform incision (depending upon whether the lesion is situated in the middle or upon the margin of the tongue, upon the inner surface of the lip or upon the red margin) should be carried well into the deeper tissues.

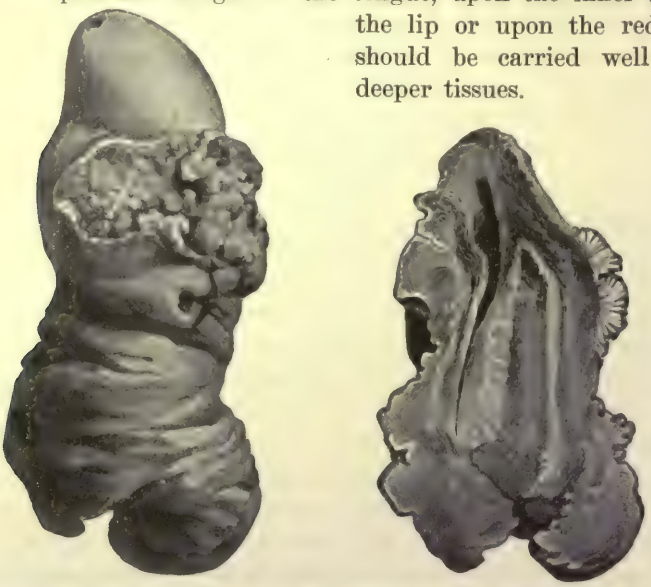


FIG. 424.—PAPILLARY CARCINOMA OF THE CORONA GLANDIS AND PREPUCE.

[We have no method of curing leucoplakia at the present time. If the epithelium affected is removed a leucoplakia reforms when the epithelium regenerates.]

Squamous-cell carcinomas of the mucous membranes appear in forms very similar to those of their companions in the skin. The deep carcinomas with craterlike, deeply fissured ulcers which have raised, often nodular, edges and an extensively infiltrated base occur most frequently upon the tongue and the lower lip.

The papillary forms, associated with superficial and deep ulcers, occur in the intestinal mucous membranes, in the mucous membranes of the cheek, jaws, pharynx and larynx, the urinary bladder, the pelvis of the kidney, and especially upon the prepuce and glans penis. The cells composing the carcinomas of the prepuce and glans have a marked tendency to undergo cornification.

FIG. 425.—NODULAR CARCINOMA OF THE MAXILLA (woman forty-two years of age). Healed by total resection.

The nodular infiltration, the boundaries of which are indistinct, is covered by normal mucous membrane in the beginning. Later ulceration occurs, nodular growths project above the surface of the ulcer, and the new growth rapidly invades surrounding structures. A papillary carcinoma involving secondarily the branches of the trigeminal nerve may cause excruciating pain which can scarcely be controlled by morphin.

Carcinomas of the mucous membranes bleed oftener and undergo putrefactive changes much earlier than those of the skin. Hæmorrhage may be the first symptom of a carcinoma of the bladder or of the pelvis of the kidney. The discharge of a foul-smelling secretion, which occurs especially with carcinomas of the mouth, may render it almost impossible for one to remain near the patient. The swallowing of this secretion may cause severe digestive disturbances and diarrhea; aspiration may cause bronchopneumonia.

Stenosis of the larynx and œsophagus follows the contraction or proliferation associated with these new growths occurring in these organs. Sudden dyspnœa may follow inflammation of the mucous membrane surrounding an ulcer of the larynx. The swelling of the mucous membrane surrounding a carcinoma of the œsophagus may be great enough to prevent even the swallowing of liquids.

Carcinomas Developing in Mucous Membranes with Cylindrical Epithelium.—The epithelial downgrowths developing in carcinomas

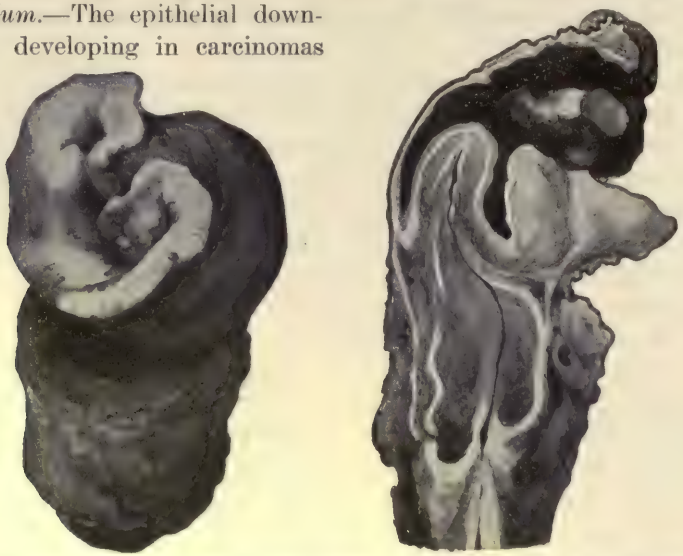


FIG. 426.—DEEP CARCINOMA OF THE PENIS WITH DESTRUCTION OF THE GLANS.

arising from mucous membranes covered with cylindrical epithelium have a glandlike or tubular form, and are lined with stratified, more rarely with simple cylindrical epithelium.

FIG. 427.—CARCINOMA OF THE LOWER LIP WHICH HAS BECOME NODULAR FOLLOWING ULCERATION. The tumor is adherent to the mandible and is fused with the submaxillary lymph nodes.

The processes in such a tumor branch frequently, communicating with each other, and the histological picture varies, as the processes may be cut transversely, obliquely, or longitudinally.

Goblet cells may be found in large numbers. They are rarely evenly distributed as they are in glands. Groups of these cells may be irregularly distributed throughout the section.

Some of the carcinomas developing from glandular epithelium have a similar tubular structure, while in

others the processes which invade the surrounding tissues are solid. The division of glandular carcinomas into the adenomatous and solid varieties is therefore justifiable.

Colloid Carcinomas.—The colloid carcinomas (mucoid) in which there is the formation of considerable mucus is another variety of this form of carcinoma.

The cells composing a colloid carcinoma are filled with mucus, and appear as large, round, swollen structures, which, because of the lateral position of their nuclei, have been called the "seal-ring cells" (Ribbert). The mucous masses which are secreted either fill the alveoli or separate the cells from the thin connective-tissue bands of the stroma.

A colloid carcinoma is composed of a soft, glassy, translucent tissue from which a viscous substance is discharged when the tumor is sectioned or scraped with a knife.

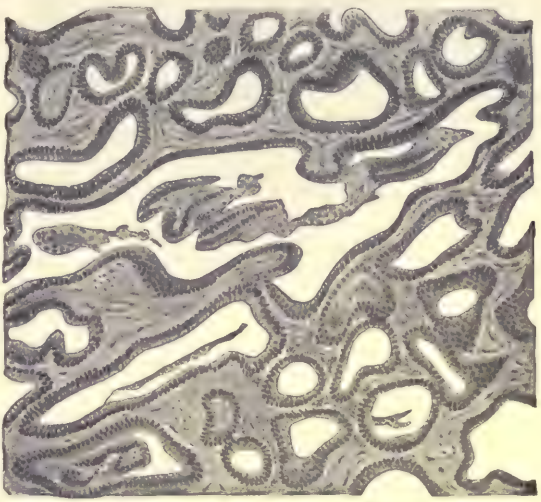


FIG. 428.—CYLINDRICAL CELL CARCINOMA OF THE RECTUM.

Most Common Sites for Development.—The cylindrical and glandular carcinomas of the mucous membranes occur most frequently in the gastro-intestinal tract, most commonly at the pylorus and on the lesser curvature of the stomach, in the cæcum, at the hepatic and splenic flexures of the colon, and in the rectum.

These tumors may also develop in the nasal mucous membrane, respiratory passages, the gall bladder, the cervix and the body of the uterus; also from the epithelial remains of the branchial clefts (branchiogenic carcinoma) and from mixed tumors. Ulcers of the mucous membrane (ulcer of the stomach) and fibroepithelial tumors (multiple intestinal polypi) form with about equal frequency the base from which these carcinomas develop.

Clinical Course.—These carcinomas begin as hard, nodular, infiltrated areas beneath the mucous membrane, apparently at first well delimited. Regressive changes soon occur, resulting in the formation of superficial and deep ulcers which in the intestine and at the pylorus are surrounded by hard raised borders, and usually tend to surround the lumen

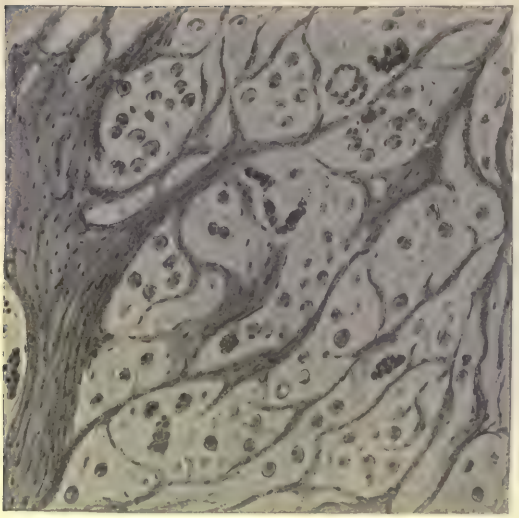


FIG. 429.—COLLOID CARCINOMA OF THE RECTUM. Section of specimen represented in Fig. 431.

of the bowel or pylorus respectively. If the nodular, cauliflowerlike, or papillary growths predominate, the lumen may be completely occluded. The cicatrizing form of carcinomatous ulcer encircling the bowel may also produce a stenosis of the bowel, sometimes so reducing the lumen that it is impossible to pass a probe or a grooved director through it. Ulceration, hæmorrhage, and putrefactive changes develop especially rapidly in carcinomas of the gastrointestinal tract, as they are exposed to the continual irritation of the intestinal contents. They are associated, depending upon the position of the tumor, with the vomiting of blood, the discharge of blood-stained mucus, and the usual symptoms

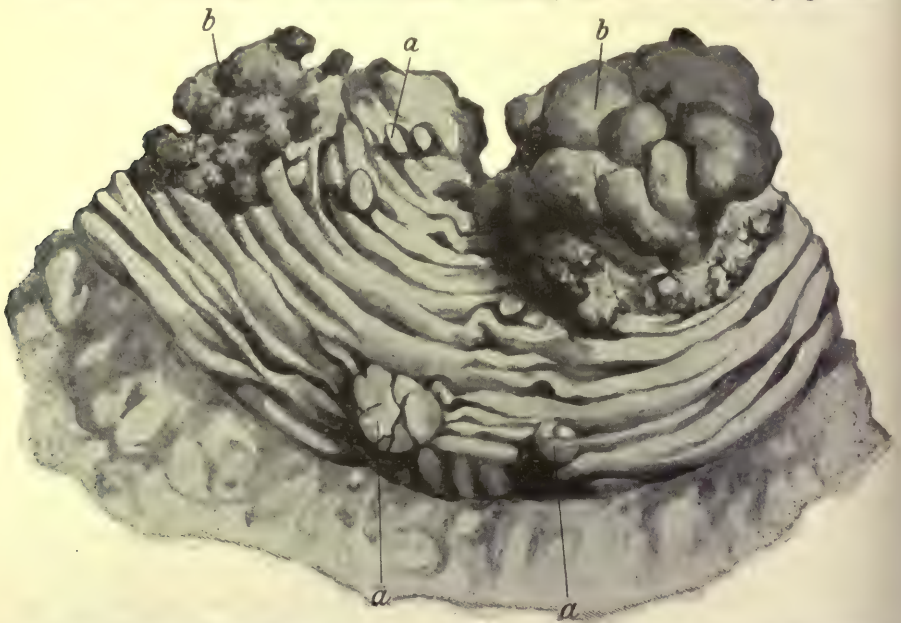


FIG. 430.—NODULAR AND PAPILLOMATOUS CARCINOMA OF THE RECTUM (*b*). *a*, MULTIPLE PAPILLOMAS. RESECTION PREPARATION.

of intestinal catarrh caused by the passage of putrefying masses over the mucous membrane.

Many of these tumors infiltrate rapidly and extensively the walls of the stomach and intestines without ulcerating. The part involved then becomes transformed for a considerable extent into a rigid, tubelike structure, the walls of which are thickened and lined by a nodular mucous membrane which cannot be displaced upon the subjacent tissue. The development of small nodules and fine strands in the serosa indicate lymphatic involvement.

If a carcinoma involving the stomach or intestine extends through their walls and ruptures into the peritoneal cavity, a general or local putrefactive peritonitis follows, depending upon whether or not adhesions have formed. An intestinal loop or a part of the stomach involved by a carcinoma may contract adhesions with a neighboring viscus, and then the latter becomes involved. Adhesions may be contracted with an intestinal loop or some hollow viscus, such as the bladder, and when ulceration and perforation occur a communication is established between the two.

FIG. 431.—COLLOID CARCINOMA OF THE RECTUM REMOVED FROM A YOUNG WOMAN TWENTY-THREE YEARS OF AGE.

Colloid carcinomas are found most frequently in the rectum, but they also occur in the stomach and cæcum (Fig. 431). They form large growths, invade wide areas, and have a tendency to progressive infiltration of the intestinal wall, the mesentery, omentum, the appendices epiploicæ, and the entire peritoneum.

Importance of Early Diagnosis.—It is essential that an early diagnosis be made in these cases, in order that efficient treatment may be instituted. This is often difficult, as the physical signs and symptoms are not pronounced, and the latter, even when marked, frequently resemble closely those occurring in other diseases. Therefore it is frequently the case that a positive diagnosis is made when the carcinoma is too far advanced for radical removal.

Carcinomas developing in the mouth are naturally noted early. They are, however, often mistaken for syphilitic lesions, as they are frequently

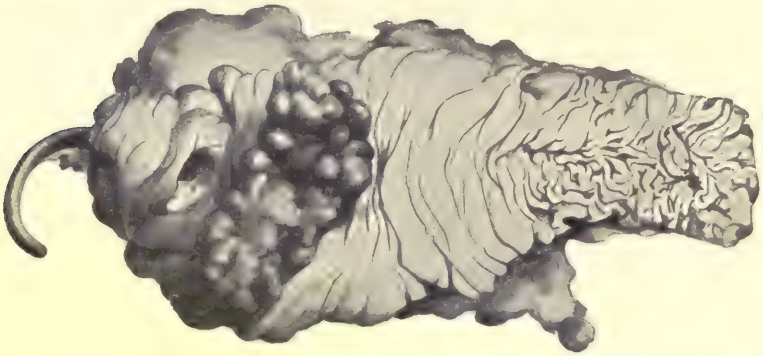


FIG. 432.—NODULAR, CIRCULAR CARCINOMA OF THE CÆCUM, THE CENTER OF WHICH IS ULCERATED. The carcinoma was situated close to the ileocæcal valve. Removed from a man thirty-five years of age. Three years have elapsed since operation with no recurrence.

surrounded by or associated with leucoplakia. If a piece of tissue is excised for microscopic examination, it should be large enough to permit of a positive diagnosis and should be taken from the proper part of the lesion.

FIG. 433.—ULCERATED SUPERFICIAL CARCINOMA OF THE RECTUM. *a*, Craterlike margin; *b*, ulcer.

Symptoms. — The symptoms depend upon the position of the tumor and upon interference with the function of the organ involved. A carcinoma of the larynx causes hoarseness; of the stomach, vomiting and chronic gastritis; of the intestines, symptoms of stenosis and chronic ileus; of the rectum, hæmorrhage and the discharge of blood-stained mucus. A tumor of the bladder is associated with hæmor-

rhage and the retention of urine; while a tumor of the antrum of Highmore may be associated with the symptoms of an empyema of the antrum, following the accumulation and subsequent infection of the secretion of the mucous membrane. The symptoms produced by a carcinoma developing in any of the organs or parts above mentioned may be confused with those associated with relatively harmless lesions.

If the tumor is visible and palpable, the diagnosis can usually be easily made, as the form of the ulcer, the induration of its edges, the appearance of its floor, the character of the secretion, and, when far advanced, the infiltration of adjacent tissues and the involvement of regional lymph nodes are quite characteristic. In carcinomas involving

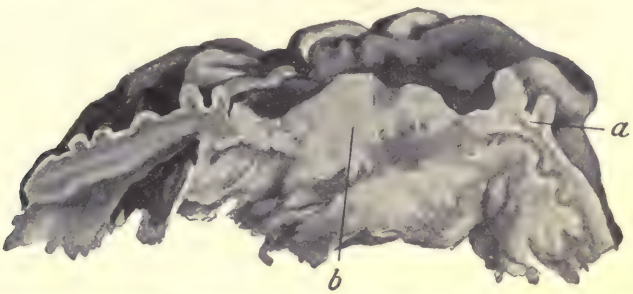


FIG. 434.—SECTION THROUGH CARCINOMA REPRESENTED IN FIG. 433. *a*, Mucous membrane at margin of the ulcer; *b*, tumor tissue.

the gastrointestinal tract it is often necessary to perform an exploratory laparotomy before a positive diagnosis can be made. If a tumor can be palpated through the abdominal wall, the diagnosis may be made, but in the majority of these cases the tumor has then extended so far that radical removal is impossible. It is advisable, when the symptoms indicate a carcinoma of the gastrointestinal tract, to perform an exploratory laparotomy, in order that an early and positive diagnosis may be made.

Treatment.—If the diagnosis is made early enough to permit of operative procedures, the tumor and the lymph nodes which drain it should be removed.

(c) CARCINOMAS OF GLANDULAR ORGANS

Histology.—Carcinomas developing in the various glands correspond more or less closely, histologically, to the glands in which they occur, and therefore differ widely in their structure. Usually an adenomatous can be differentiated from a solid type, depending upon whether the cells are arranged in the form of gland tubules or solid processes. The epithelial cells may be cylindrical, cubical, or polymorphous, and arranged in a single layer or stratified. The secretion of the cells differ,

depending upon the origin of the tumor. The cylindrical cells in a carcinoma of the thyroid may secrete colloid, while those found in a primary carcinoma of the liver may secrete bile. The cylindrical cells found in carcinoma of the breast may, like those found in adenocarcinoma of the gastrointestinal tract, secrete mucus.

If the gland tubules become dilated as the result of the accumulation of the secretion, small and large cysts may be formed which communicate with each other. The epithelium lining these cysts may proliferate to form large papillary growths which completely fill the cavity of the cyst (papillary cystocarcinomas of the breast and ovary).

Mucoid degeneration leads to the formation of a colloid carcinoma (e. g., in the breast), just as in cylindrical-cell carcinomas.

Predisposing Causes.—Chronic inflammation, contusions, and benign growths (e. g., fibroadenomas of the breast) are regarded by many as predisposing causes. The etiological relations, however, between the lesions above mentioned and malignant growths are based upon clinical observations confined almost entirely to the female breast.

Appearance of These Growths.—The clinical appearance of the carcinomas occurring in the different glands are very similar. They differ in consistency, the scirrhus forms being hard, the medullary forms soft. The tumors develop as round nodules which invade the normal tissues in all directions and replace them. The nodules become fused with the adjacent tissues. The carcinoma extends, when it reaches the surface of an organ, to the adjacent structures (e. g., a carcinoma of the prostate may extend into the bladder and rectum). When it reaches and invades the mucous membrane or skin regressive changes soon occur, resulting in the formation of deep ulcers and nodular growths.

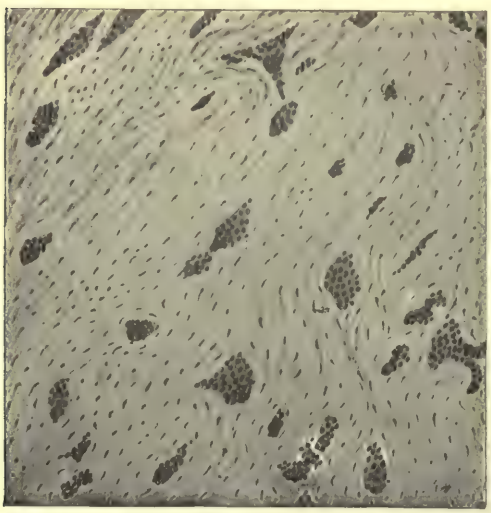


Fig. 435.—Carcinoma of the Breast (Scirrhus).

The female breast affords the best examples of carcinomas developing in glands. Usually the nodule, which develops without symptoms, is accidentally noticed. It appears as an indurated area, the size of a cherry, within the substance of the breast. The boundaries of the nodule are indistinct, and it is firmly adherent to the surrounding tis-

sue. If the tumor lies beneath the nipple or close to it, the latter will be somewhat retracted and more difficult to grasp than is normally the case, as the processes which radiate out from the new growth hold it fast. While the induration, which is usually associated with radiating pains, enlarges, the skin covering it becomes dark red in color, and

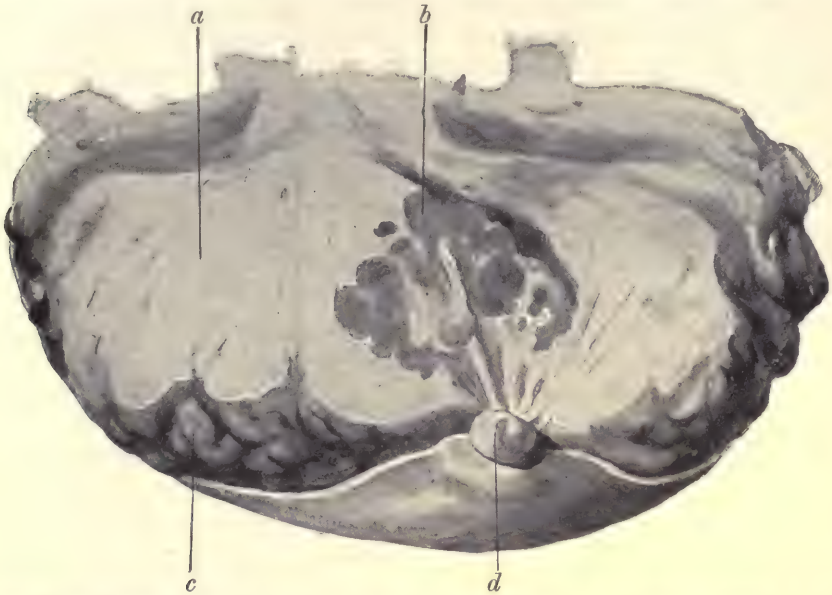


FIG. 436.—SECTION THROUGH A CARCINOMA OF THE BREAST. *a*, Mammary gland; *b*, carcinoma; *c*, subcutaneous fat; *d*, retreated nipple which is adherent to the new growth.

nodules may develop in it and processes may extend into the subjacent fascia, muscles, and even to the thorax, so that finally the tumor, which in the beginning could be displaced with the breast, becomes firmly attached and can no longer be displaced upon the pectoral muscles or ribs. The infiltrated skin ulcerates and the tumor grows to form a large mass which includes all of the breast (medullary form), or contracts to form a nodular mass adherent to the chest wall, which undergoes here and there regressive changes, ending in ulcer formation (scirrhus). Enlarged, indurated lymph nodes are usually found early in the disease and may be palpated in the axillary fossa. Considerable hæmorrhage may occur from the ulcers, from the intercostal and internal mammary arteries. Hæmatogenous metastases develop in the lungs, the liver and other viscera, and frequently in the bones. Death finally occurs as a result of an increasing cachexia or a severe hæmorrhage.

The clinical symptoms of carcinomas developing in glandular organs naturally depend upon the position of the growth. The symptoms belong to special surgery.

(d) THE CLINICAL COURSE OF CARCINOMA

The clinical course is very variable. Local invasion and destruction of tissue, the development of lymphogenous and hæmatogenous metastases, and cachexia are common to all. But sometimes the clinical course is slow, at other times relatively rapid. Varying energy of growth, the location of the tumor, and the formation of metastases are important factors in determining how rapidly or slowly the disease will progress. The causes of death are as different as the courses of the disease. If death does not occur from some secondary disease, such as perforative peritonitis or meningitis, aspiration pneumonia or hæmorrhage, it is usually caused by exhaustion.

Carcinomatous Cachexia.—After a shorter or longer period the condition of the patient undergoes very significant changes. Rapid and marked emaciation, loss of strength, and anæmia combine to produce the picture of carcinomatous cachexia, which is partly due to the absorption of products of decomposition from the tumor, and partly to secondary disturbances (repeated hæmorrhages, interference with the taking of food, etc.) and to the development of metastases in important viscera.

Prognosis.—The prognosis of cancer is very unfavorable, but varies with the different forms. The superficial carcinomas of the skin are relatively benign, as they grow slowly and at first are localized, forming metastases late. The soft, rapidly infiltrating forms, especially the colloid carcinomas of the viscera and mucous membranes, belong to the worst forms. Carcinomas developing in young and middle-aged adults are usually more malignant than those developing in old people. The absolute decrease in the number of lymphatics which occurs with age may account for the lessened malignancy of these growths in old people.

Duration.—Usually the clinical course is of from two to three years' duration. The superficial carcinomas of the skin and the contracting, cicatrizing forms are the only ones which have a longer course.

Cures.—A permanent cure can only be expected when a radical operation is performed early. Statistics concerning permanent cures following radical operations differ widely. The differences in statistics being due to the position of the tumor, the extent of the operation required, the technic employed, the skill of the operator, and whether the case came to operation early or late. Statistics are, however, encouraging, as they show that the relative number of cures of at least three years' duration have steadily increased with improvement in diagnostic methods and technic. Even if there are recurrences after radical operations, the length of life of the patient is considerably increased by such a procedure.

Radical Removal.—Operative treatment is suited only for those cases in which the carcinoma can be completely removed without danger to

life. The extent of the primary tumor, the amount of lymphatic involvement, and the presence of or suspicion of hæmatogenous metastases should be considered before a radical operation is undertaken. Clinical experience has shown that even a small carcinoma of the breast is inoperable when, in addition to the involvement of the axillary lymph nodes, there is involvement of the supraclavicular nodes, for it has been demonstrated that when the lymphogenous metastases are so extensive, recurrences are rapid, even after radical removal of the breast and the involved nodes (von Bergmann). This statement has been substantiated by the later investigations of Küttner in von Bruns's clinic. The general condition of the patient should also be carefully considered before an operation is undertaken. As a rule, an operation should not be undertaken if the tumor has already reached such a size that it threatens life.

Recurrences.—Recurrences following operations are more frequent in the tissues surrounding the extirpated lymph nodes than in those about the primary tumor. This is easily explained, for in spite of the removal of the enlarged lymph nodes, together with the fat and fascia surrounding them, small, imperceptible lymph nodes may be left behind which already contain carcinoma cells. Local recurrences follow most frequently those carcinomas which send out continuous or interrupted processes into the lymphatic vessels, which extend well beyond the apparent boundaries of the primary tumor. Recurrences about the scar, which occur after long intervals, may be regarded as new tumors, especially if there are preëxisting lesions (e. g., a leucoplakia) which favor the development of carcinoma. Nothing definite can be said concerning the so-called *implantation recurrences* which are supposed to follow the implantation of carcinoma cells in the wound during an operation. In these cases the transmission of carcinoma cells through the lymphatics must be definitely excluded before an opinion can be given.

Causes of Carcinoma.—Nothing definite is known concerning the cause of carcinoma. The principal theories have already been discussed in the general discussion of tumors.

The fact that carcinomas not infrequently develop from wounds and scars, from benign new growths and chronically inflamed tissues is used by the defendants of Virchow's theory of chronic irritation to show that the latter may produce changes in the cells which result in unrestricted proliferation. Congenital carcinomas and the development of a carcinoma from demonstrable embryonal rests, both of which are rare, also the occurrence of a squamous-cell carcinoma in mucous membranes composed of columnar cells have been used to substantiate Cohnheim's theory that tumors developed from displaced embryonal rests.

According to Ribbert, the general cause of carcinoma formation is the displacement of small islands of epithelium from their normal con-

nections, the displacement following the proliferation of the connective tissue, which may be caused by a number of different agents.

The parasitic theory concerning the origin of carcinoma has won many friends. This theory rests partly upon an analogy between diseases which are undoubtedly of an infectious origin and carcinomas, and partly upon clinical observations that carcinomas occur most frequently upon parts exposed to irritation (e. g., about the orifices of the body, in narrow or tortuous parts of the gastrointestinal tract), in ulcers or chronically inflamed tissues, in man and wife or many members of the same family. These facts observed clinically may be most easily explained by assuming an infectious origin. But the structures which have been regarded as parasites have been proven by later investigations to have been inclusions of epithelial cells or leucocytes. The peculiar and different appearance of the cell-inclusions are dependent upon the extent of the regressive changes in the latter. The microorganisms which have been found and cultivated cannot be regarded as the cause of carcinoma. Besides, the histology of certain forms of carcinoma and the way in which they develop are important arguments against the parasitic theory.

Heredity.—The question of heredity is still unsettled, as it is difficult to estimate the value and accuracy of statistics regarding it. Clinically, it is quite striking that many members of the same family may apparently inherit the tendency to carcinoma formation and that the same organ may be primarily involved.

LITERATURE.—Ausführliche Literaturangaben bei *Borst.* Die Lehre von den Geschwülsten. Wiesbaden, 1902, II, p. 966.—*v. Bergmann.* Das lupusähnliche Karzinom. Handb. der prakt. Chir., 2. Aufl., Bd. 1, p. 47.—*Borrmann.* Das Wachstum und die Verbeitungsweise des Magenkarzinoms. Kitteil. aus d. Grenzgeb., Bd. 1, Suppl., 1901;—Die Entstehung und das Wachstum des Hautkarzinoms, nebst Bemerkungen über die Entstehung der Geschwülste im allgemeinen. Zeitschr. f. Krebsforsh., Bd. 2, 1904;—Statistik und Kasuistik über 290 histol. untersuchte Hautkarzinome. Deutsche Zeitschr. f. Chir., Bd. 76, 1905, p. 404.—*Coenen.* Zur Kasuistik und Histologie des Hautkrebses. Arch. f. klin. Chir., Bd. 78, 1905, p. 801.—*Hauser.* Das Zylinderepithelkarzinom des Magens und des Dickdarms. Jena, 1890.—*Heimann.* Die Verbreitungsweise der Krebserkrankung. Arch. f. klin. Chir., Bd. 57, 1898, p. 911.—*Krompecher.* Der Basalzellenkrebs. Jena, 1903.—*Küttner.* Welche Aussichten bietet die Operation des Mammakarzinoms beit vergrösserten Supraklavikulardrüsen? Beitr. z. klin. Chir., Bd. 36, 1902, p. 531.—*Milner.* Gibt es "Impfkarzinome"? Arch. f. klin. Chir., Bd. 74, 1904, p. 669.—*Fr. Müller.* Stoffwechseluntersuchungen bei Krebskranken. Zeitschr. f. klin. Med., Bd. 16, 1889, p. 496.—*Orth.* Die Morphologie der Krebse und die parasitäre Theorie. Berl. klin. Wochenschr., 1905, p. 281.—*Petersen.* Beiträge zur Lehre vom Karzinom. Beitr. z. klin. Chir., Bd. 32, 1902, p. 543;—Ueber Heilungsvorgänge im Karzinom (Riesenzellen). *Ibid.*, Bd. 34, 1902, p. 682.—*Ribbert.* Geschwulstlehre. Bonn, 1904, p. 459;—Die Entstehung des Karzinoms. Bonn, 1905;—Beitr. zur Entstehung d. Geschwülste. Bonn, 1906.—*v. Volkmann.* Ueber den primären Krebs der Extremitäten. v. Volkmann's Samml. klin. Vortr., 1889, Nos. 334–335.

E. ENDOTHELIAL TUMORS

ENDOTHELIOMAS

Von Recklinghausen (1862) was first to recognize that endothelial cells composed the proliferating part of these tumors. This fact has been especially emphasized by Köster. The classification of these tumors, in spite of the amount of investigation that has been devoted to them, is still a mooted question.

Nature of Endothelium.—The intimate relationship between endothelium and connective tissue (His) does not permit one to draw a sharp-and-fast line of distinction between these tumors and sarcomas. Histologically, endotheliomas resemble some epitheliomas so closely, especially those in which there is the formation of gland tubules, that it is impossible to give any opinion acceptable to all pathologists as to the nature of the cells (e. g., Krompecher's carcinoma basocellulare, p. 938; mixed tumors of the salivary glands, p. 971; cholesteatomas, cylindromas). Besides, there is no uniformity of opinion among embryologists and histologists as to the classification of endothelium, the latter being regarded by some as connective tissue, by others as epithelium. Clinically these tumors have certain peculiarities.

It is impossible to make a positive clinical diagnosis of an endothelioma. The microscope must decide the nature of the tumor. Even an experienced pathologist may have great difficulty in making a microscopic diagnosis and differentiating a tumor of this character from certain forms of carcinoma, sarcoma, and adenoma.

It is customary to divide these tumors into *lymphangio-endotheliomas, hæmangio-endotheliomas*, and into special forms, such as *peritheliomas*, and *endotheliomas* of the *dura mater* (psammomas).

Lymphangio-endotheliomas.—Lymphangio-endotheliomas develop from the endothelium lining the tissue spaces and lymphatic vessels. These tumors are composed of columns of cells which correspond to the course of the lymphatics. The cells, which have a tubular or solid arrangement, are flat, cubical, or columnar. The three varieties may be found in different parts of the tumor or combined in the same field. The cell columns are enlarged at the nodal points, and the cells composing them may be concentrically arranged. According to Borst the cell columns, which differ in size, have a delicate linear arrangement and are lined by two layers or a single layer of cells, resembling very closely the histological picture presented by proliferating lymphatic vessels. These tubules may dilate to form cysts. If papillary growths then develop,

the tumor may be mistaken for a cavernous or cystic lymphangioma or for a papillary cystadenoma (e. g., of the sweat glands).

Stroma.—The character of the connective-tissue stroma varies. It may be very cellular, mucoid, or fibrous. Probably a metaplasia of the stroma into cartilage, occasionally even into bone, occurs, the presence of cartilage and bone in mixed tumors of the parotid and related forms of endotheliomas being explained in this way (p. 974).

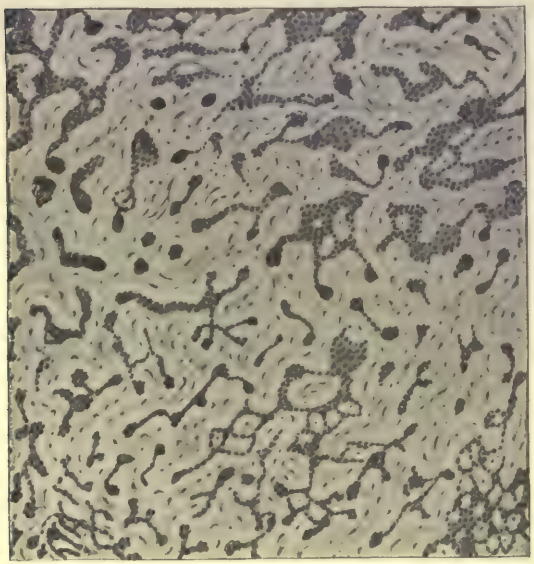


Fig. 437.—Lymphangio-endothelioma of the Skin.

Most Common Sites for Development. — *Lymphangio-endotheliomas* of the skin and subcutaneous tissues, especially of the face, appear as circumscribed, encapsulated nodules or non-encapsulated growths which are sometimes regarded as sarcomas, sometimes, especially when ulcerated, as carcinomas of the skin. They grow slowly, but rarely forming metastases, and therefore are to be regarded as relatively benign tumors. According to Tanaka, the lymph nodes when involved are of a soft, fluctuating consistency, and are not adherent to the surrounding tissues.

Similar tumors, which have been regarded as lymphangio-endotheliomas, occur in the different viscera, in the membranes of the central nervous system, in the bones, the ovary and testicle, and in the salivary glands. The latter are sometimes regarded as mixed tumors (Wilms), and sometimes as fibroepithelial growths (Ribbert). As a result the group of endotheliomas has been considerably reduced. These tumors are usually encapsulated.

Endotheliomas of Pleura and Peritoneum.—Endotheliomas of the pleura and peritoneum are rare. They produce a diffuse, thick infiltration of the membrane involved, and frequently form metastases. Endotheliomas of the pleura may also produce large nodular tumors which invade the lung. Depending upon the view held concerning the origin and nature of the cells covering the serous membranes, these tumors are classified as endotheliomas or carcinomas.

Hæmangio-endotheliomas. — Hæmangio-endotheliomas develop from the endothelium of blood vessels.

The proliferating capillaries and small vessels which compose these tumors are lined or filled with tall, actively proliferating endothelial cells. They appear upon microscopic examination as glandlike tubules or solid cell columns which branch repeatedly and communicate with each other. If the proliferating endothelium is cubical or cylindrical, the tumor can only be differentiated from a new growth developing from a gland by the presence of blood in the lumina of the vessels.

Hæmangio-endotheliomas usually grow slowly, are circumscribed, and have but little tendency to invade the surrounding tissues and form metastases. They have been observed in the different viscera and in the bones, occurring in the latter also as multiple growths (Fritz König, Narath). These tumors resemble upon section hæmangiomas or very vascular, soft sarcomas. Endotheliomas occurring in bone produce a pressure atrophy and expansion of the cortex. They are so vascular that they often give rise to symptoms resembling those of an aneurysm.

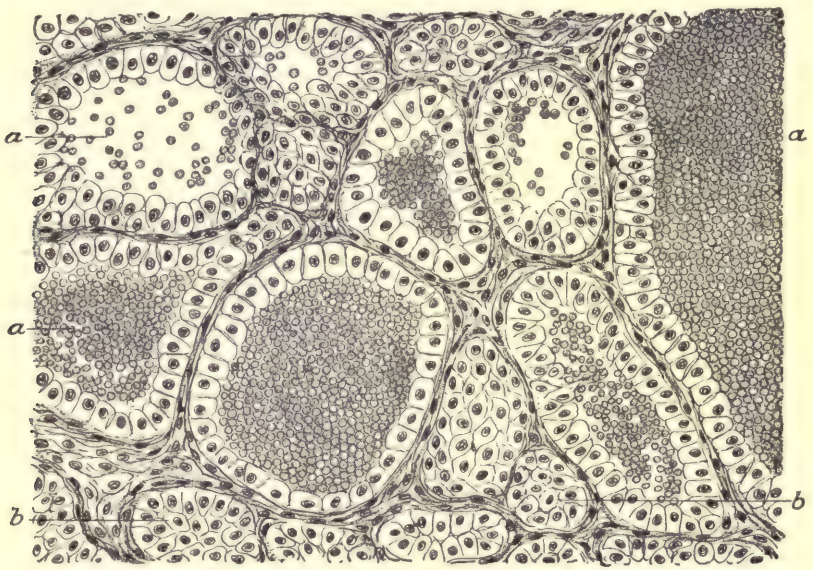


FIG. 438.—HÆMANGIO-ENDOTHELIOMA OF THE KIDNEY. *a*, Blood vessels containing blood; *b*, blood vessels filled with proliferating endothelial cells. (From Ziegler's General Pathology.)

Peritheliomas.—Peritheliomas are a variety of hæmangio-endotheliomas; sometimes they are regarded as lymphangio-endotheliomas.

These tumors may develop from the adventitial cells of the blood vessels, which apparently are very similar to endothelial cells, or from the endothelium of the perivascular lymphatics. The tumor tissue is

composed of dilated capillaries, which are surrounded by a wide zone of cells of different forms. The cell mantles, or, more correctly, the cell cylinders, containing the blood vessels are very sharply differentiated from the poorly developed connective-tissue stroma.

The vessels surrounded by the cells have a cirsoid arrangement, branch frequently, and give rise to a very characteristic histological pic-

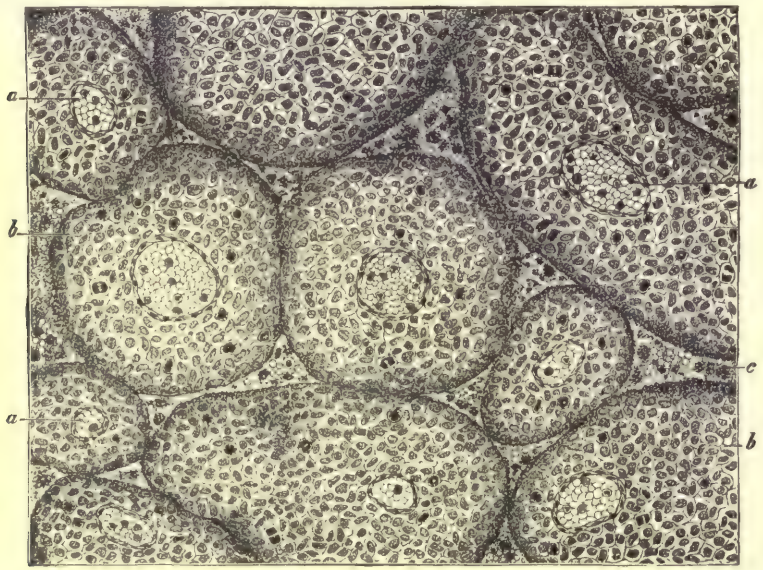


FIG. 439.—PERITHELIOMA OF THE THYROID GLAND. *a*, Section through a vessel; *b*, perivascular cylindrical cells with many mitoses; *c*, granular masses and cells between the cell columns. (From Ziegler's General Pathology.)

ture. The latter is so characteristic that Waldeyer has called these tumors plexiform angiosarcomas; Kolaczek, angiosarcomas. It seems best to avoid the use of these terms, however, as the tumors might then be confused with very vascular sarcomas, which might also be called angiosarcomas, or, better, telangiectatic or cavernous sarcomas (Borst).

Sarcomas in which the proliferation of perivascular cells is pronounced resemble peritheliomas very closely, when the cell cylinders are fused. If hyaline degeneration occurs and the vessels become obliterated, the tumor resembles a cylindroma.

Peritheliomas are found most frequently in the brain and in the membranes surrounding it. They appear as circumscribed nodules or as diffuse, even multiple, infiltrations. They are also found in the subcutaneous tissue, especially in the subcutaneous tissues of the cheeks and lower lip, in the bones, muscles, and different viscera. The encapsulated

tumors developing from the carotid gland and situated at the bifurcation of the common carotid are usually peritheliomas.

Peritheliomas are relatively benign tumors.

Psammomas.—Tumors of the dura mater form a special class of endotheliomas. They contain small calcium granules which resemble sand, such as normally occurs in the pineal gland and upon the inner surface of the dura. The term psammoma was applied to these tumors by Virchow.

These tumors develop from the endothelial cells covering the inner surface of the dura, and appear as grayish red, circumscribed, firm,

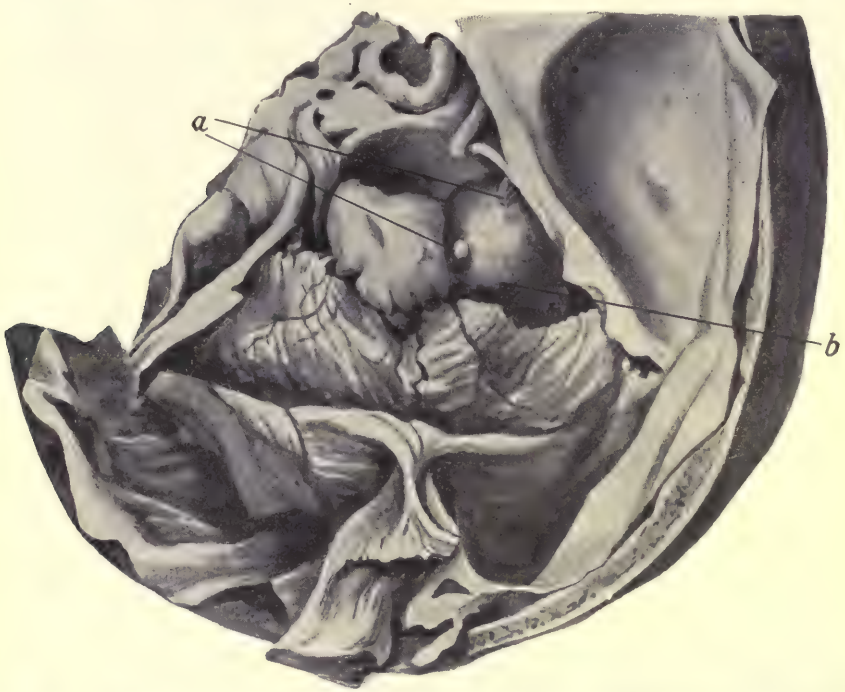


FIG. 440.—PSAMMOMA OF THE DURA SITUATED UPON THE POSTERIOR SURFACE OF THE RIGHT PETROUS BONE AND PRODUCING A DEPRESSION IN THE FLOCCULUS, THE CORRESPONDING SUPERIOR CEREBELLAR PEDUNCLE AND THE PONS. The trunk of the fifth nerve (*a*) runs through the tumor mass (*b*). The facial and auditory nerves lie upon the outer side of the tumor and are fused with its capsule. The only symptoms produced by this tumor, which was almost as large as a walnut, was severe trifacial neuralgia. The patient, a woman seventy-three years of age, died following the removal of the Gasserian ganglion and the tumor was found during the post-mortem examination. (Lexer.)

hemispherical tumors. They are attached to the dura by a broad base or short pedicle, and produce a depression in the surface of the brain, being separated from the latter by a vascular capsule. They usually occur as single, more rarely as multiple growths, and are found more

frequently upon the upper than upon the lower surface of the brain. These tumors differ in size. Only the larger tumors, the size of a walnut or apple, produce symptoms, which, of course, are those of a brain tumor. The smaller tumors, often no larger than a pinhead, produce no symptoms and are usually accidentally discovered during post-mortem examinations. Usually they grow slowly. The cellular forms may develop relatively rapidly and rupture through the dura mater and bone.

These tumors do not form metastases. The dangers associated with them depend altogether upon the position of the tumor.

The relation between the parenchyma and stroma varies in different tumors. The cells composing the former are flat or polymorphous and are arranged in groups or columns. These groups or columns are surrounded by connective-tissue trabeculæ which contain few or many cells. Among the endothelial elements, which under the microscope appear in well-defined alveoli or columns, may be seen varying numbers of stratified bodies composed of cells concentrically arranged. These undergo calcification and become transformed in the white bodies resembling grains of sand. Connective-tissue bundles may also undergo hyaline degeneration and calcification.

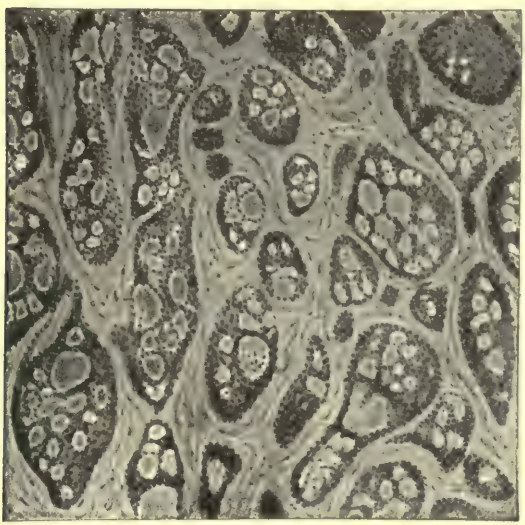


Fig. 441.—Cylindroma of the Orbit.

Similar tumors also occur within the orbit, developing in the dural sheath of the optic nerve (Ribbert). They are also found in the pia mater, the pineal gland, and the choroid plexus. Multiple tumors of this character are occasionally found in the peritoneum (Borst).

Cylindromas.—The cylindromas, first described by Billroth in 1856, and regarded by Köster as endothelial growths with hyaline degeneration of the cell columns, are classified by many authors with endotheliomas.

The small, glassy, hyaline bodies of round, bulbous, cordlike or cylindrical form with numerous branches and bulbous expansions, which may be easily isolated from the cut surface of a fresh tumor, are character-

istic. The histological picture is quite characteristic, as these glasslike bodies are surrounded by a broad mantle of cells. If there are no cell rests or fibrillæ within the bodies above mentioned, they appear as lumina of large vessels.

A similar histological picture may be produced in a number of different forms of tumors by hyaline degeneration of the cells or by secretion poured out from them. For these reasons a number of pathologists (Ziegler, Orth, Lubarsch) are unwilling to place cylindromas in a separate and distinct class.

A similar histological picture is presented by vascular sarcomas after hyaline degeneration of the cells and obliteration of the lumina of the vessels, by adenomas and carcinomas after the secretion of a hyaline material and its accumulation in the glandlike tubules, or between the cells forming a solid column. One can, therefore, speak of a sarcoma, carcinoma, or adenoma cylindromatosum.

The changes above described occur most frequently in endotheliomas and peritheliomas. Borst regards these two forms as true cylindromas. Ribbert also places them in a separate group, but regards them not as endothelial new growths, but as fibroepithelial tumors, believing that they develop from mucous or closely allied glands.

These tumors have a slow growth, are encapsulated, and often may be recognized upon gross examination by the cavities containing hyaline masses. They rarely invade the surrounding tissues, and are rarely followed by metastases. They may be regarded as benign tumors. The hyaline changes occur partly within the endothelial, or, according to Ribbert, epithelial cell columns, partly within the connective-tissue stroma lying between them.

These tumors develop most frequently in the orbit, the salivary glands, the palate, the floor of the mouth, the antrum of Highmore, and the nose. Sometimes they are found in the skin, the membranes of the brain and cord, in the peritoneum, in muscles and bone.

They have no characteristics which make a positive clinical diagnosis possible. Developing in the salivary glands or palate, they resemble closely, clinically, encapsulated adenomas or mixed tumors. If they extend from the orbit into the nose, the antrum of Highmore, or the frontal sinus, they may resemble a carcinoma or sarcoma.

Treatment.—The treatment of all the tumors of the endothelial group consists of removal. If they are encapsulated they may be enucleated. If they have no capsule and have invaded the neighboring tissues, they should be treated as malignant tumors.

LITERATURE.—*Billroth.* Untersuchungen über die Entwicklung der Blutgefässe nebst Beobachtungen aus der Berliner Klinik. Berlin, 1856, Die Zylindergeschwulst, p. 55.—*Borst.* Die Lehre von den Geschwülsten. Wiesbaden, 1902, II, p. 953.—

Burkhardt. Sarkome und Endotheliome nach ihrem path.-anat. und klin. Verhalten. Beitr. z. klin. Chir., Bd. 36, 1902, p. 1.—*Hildebrand.* Ueber Resektion des Penis wegen eines Endothelioma intravasculare. Deutsche Zeitschr. f. Chir., Bd. 48, 1898, p. 209.—*Hinsberg.* Die klinische Bedeutung der Endotheliome der Gesichtshaut. Beitr. z. klin. Chir., Bd. 24, 1899, p. 275.—*Fritz König.* Ueber multiple Angiosarkome. Arch. f. klin. Chir., Bd. 59, 1899, p. 600.—*Köster.* Kankroid mit hyaliner Degeneration (Cylindroma Billroths). Virch. Arch., Bd. 40, 1867, p. 468.—*Mulert.* Ein Fall von multiplen Endotheliomen der Kopfhaut. I.-D., Rostock, 1897.—*Narath.* Pulsierendes Angioendotheliom des Fusses. Chir.-Kongr. Verhandl., 1895, II, p. 427.—*Nasse.* Die Geschwülste der Spiecheldrüsen und verwandte Tumoren des Kopfes. Arch. f. klin. Chir., Bd. 44, 1892, p. 233.—*v. Recklinghausen.* v. Gräfes Archiv für Ophthalmologie, Bd. 10, Abt. 2, 1864, p. 62.—*Ribbert.* Geschwulstlehre. Bonn, 1904.—*Tanaka.* Ueber die klinische Diagnose von Endotheliomen und ihre eigentümliche Metastasenbildung. Deutsche Zeitschr. f. Chir., Bd. 51, 1899, p. 209.—*Volkmann.* Ueber endotheliale Geschwülste, zugleich ein Beitrag zu den Speicheldrüsen- und Gaumentumoren. Deutsch. Zeitschr. f. Chir., Bd. 41, 1895, p. 1.

F. MIXED TUMORS

Definition.—By the term mixed tumor is usually understood a tumor which is composed of different tissues. They are distinguished from the combined forms of connective-tissue tumors, such as fibrolipomas, osteochondrosarcomas, lymphangiofibromas, etc., and also from the fibroepithelial growths in which the epithelium resembles mucous membrane, skin, or glandular epithelium.

Mixed tumors form a distinct group which vary a great deal in their histological characteristics. As there are so many transitional forms, a division into other groups is necessary. If the classification is based upon the structure, which is sometimes simple, sometimes complicated, and at other times highly organized, the three following forms may be differentiated:

1. Simple mixed tumors of different organs.
2. Teratoid tumors with two varieties:

(*a*) Complicated dermoid cysts of the ovary and testicle (cystic embryomas of Wilms).

(*b*) Teratoid mixed tumors (embryoid tumors of Wilms).

3. Teratomas.

CHAPTER I

SIMPLE MIXED TUMORS

Nature and Origin.—These tumors, varying so much in structure, have attracted the attention of a number of investigators, but even at

the present time there is no uniformity of opinion concerning their nature and origin. Some pathologists believe that they are the result of metaplasia or of a tumorlike metamorphosis of adult tissues; while others think that they develop from displaced pieces of germinal tissue, supporting Cohnheim's theory concerning the cause of tumor formation.

The latter view appears to Wilms to be the more plausible. According to him these tumors develop from undifferentiated germinal tissue which was displaced very early in fœtal life and is potentially able to reproduce any of the tissue normally found in the part from which it was displaced.

The group of simple mixed tumors comprises tumors of the salivary glands, breast, and of the urogenital system. Some of these tumors are well known clinically. Within this group of simple tumors are some which are relatively complicated. It is impossible to make a sharp distinction between them and fibroepithelial growths on the one hand and teratoid tumors on the other.

Mixed Tumors of Salivary Glands.—Mixed tumors of the salivary glands are found most frequently in the parotid, more rarely in the other salivary glands. They appear as well-circumscribed growths which may develop at any age, being most common in the second and third decennia. These tumors grow very slowly, cases having been observed in which the growth has existed from twenty to forty-five years without enlarging to any great extent. Occasionally, however, they assume rapid growth and reach considerable size (as large as a man's head). Usually they are encapsulated and sharply defined against the surrounding tissue, are displaceable upon the subjacent tissues, and covered by normal skin, which may, however, be tense and thin if the tumor is large. The surface of such a tumor is usually nodular, the nodules

FIG. 442.—MIXED TUMOR OF THE PAROTID GLAND.

being large. The consistency is usually hard, but frequently in some parts of the tumor soft, even pseudo-fluctuating areas may be found between the firm, resistant parts.

The completely encapsulated growths lie between the lobules or upon the surface of the parotid gland, in which they produce a bed by their expansive growth. At times a tumor is attached to the gland by a pedicle. Occasionally multiple tumors are found within the salivary glands.

Position.—If the tumors develop in the anterior part of the parotid gland, they lie just in front of the ear, between the zygoma, the anterior border of the masseter muscle and mandible, while if they develop from the lower and posterior part of the gland they surround the lobule of the ear which they elevate and displace.

Symptoms.—Symptoms are produced by the larger tumors only. Most frequently the symptoms are facial paralysis and partial deafness, the latter being due to narrowing of the external auditory canal. Usually these tumors cause no pain, and it is easily understood why the majority of the patients seek surgical aid so late. On an average, patients carry these tumors eight years before they seek surgical aid, notwithstanding the great disfigurement. Surgical aid is usually sought because of facial paralysis or rapid increase in the size of the growth.

FIG. 443.—BENIGN MIXED TUMOR OF THE SOFT PALATE.

Malignant Degeneration and Metastases.—The submaxillary tumors develop in the submaxillary region. If they are situated in the median part of the gland they may project into the floor of the mouth. Slow growth, encapsulation, and mobility of these tumors indicate their benign character. A malignant change is indicated by rapid growth and by the invasion of the surrounding tissue, following the rupture of the growth through a part of its connective-tissue capsule. Küttner estimates that about eleven per cent of the mixed tumors which occur in the submaxillary gland become malignant. Lymphogenous and hæmatogenous metastases then develop as in carcinomas and sarcomas, depending upon whether the mixed tumor undergoes carcinomatous or sarcomatous degeneration, while the primary tumor becomes so extensive that it becomes inoperable and breaks through the skin, forming deep ulcers.

Diagnosis.—The diagnosis is based upon the position of the tumor, the slow growth, nodular form, uneven consistency, and encapsulation.

A mixed tumor which has undergone malignant degeneration can be differentiated from a carcinoma or sarcoma only by the previous history of a preëxisting tumor which has been noted for some time, and the uneven consistency. If the tumor is small, tuberculous lymph nodes and lipomas must also be considered in making a differential diagnosis. Cystic mixed tumors may easily be confused with retention cysts, which are not rare, especially in the parotid gland.

Treatment.—The indication for treatment is complete removal. Encapsulated tumors of the parotid gland can usually be removed without injuring the facial nerve. If the tumor is situated in the submaxillary gland, the latter should be removed with the tumor. Recurrences are rare. They may develop from portions of the tumor which were left behind during the operation or from other small tumors within the gland. If the tumor has become malignant, the dissection should be carried well into healthy tissues. No effort should be made to spare the facial nerve when a tumor which has undergone malignant changes is situated in the parotid gland. Recurrences develop early even after the most radical procedures, and the prognosis is bad if the tumor has already become malignant.

Mixed Tumors in Other Parts of the Head.—Similar tumors, but of a simpler structure, are also found adjacent to the parotid and submaxillary glands in the cheek (developing from accessory salivary glands), in the upper lip, in the skin of the nose, and finally within the orbit adjacent to the lacrymal gland. (Vide Plate I, Fig. 3, p. 884.)

Macroscopic Appearance and Histology.—Upon section mixed tumors usually have a lobulated structure, and so mottled an appearance that they can scarcely be mistaken for any other variety of tumor. Soft and hard, solid, cystic, and different colored areas are intermingled.

Microscopically epithelial-like cells and stroma, the relative proportion, form, and arrangement of which vary in different tumors, depending upon whether they are simple or complicated, are found.

The epithelial-like cells are cubical and cylindrical, and arranged in solid cords, in alveoli, in glandlike, or cystic and dilated tubules. Depending upon the character and arrangement of the cells, different areas may resemble histologically an adenoma or a carcinoma. Wilms thinks that the glandular areas in these tumors resemble somewhat, histologically, the anlage of the parotid, submaxillary, and lacrymal glands. In some of the areas, canals, cysts, and alveoli are found which are lined with flat epithelium (including basal and prickle cells and the horny layer of the skin—Hinsberg, Wilms).

The stroma consists of a fibrillar connective tissue containing elastic fibers or a cellular embryonal tissue. Myxomatous, cartilaginous and bony areas are also found in the complicated tumors. Different areas

of such a tumor may, therefore, resemble a fibroma, a spindle-cell sarcoma, a myxoma, chondroma or osteoma, depending upon the character of the stroma.

The relation between the parenchyma and stroma of these tumors varies a great deal. These differences explain the number of different terms, such as enchondromas, enchondroma mucosum, and myxomatodes, chondrosarcoma, chondroadenoma, which were earlier applied to these tumors.

Wilms's Theory as to Origin.—Wilms believes that not only fully developed tissues, but also embryonal cells and tissues in different stages of development are found in these tumors, for the glandlike tubules resemble closely the anlage of the gland in which they occur. He believes that the flat epithelium is derived from the epithelium of the mouth cavity or orbit depending upon the position of the tumor. He thinks that the mixed tumors of the salivary glands and allied tumors occurring in the palate develop from embryonal rests consisting of epithelium and mesenchyme which have remained latent for a long time. It depends upon the rate of growth and the character of the different cellular elements whether a simple or a complicated mixed tumor develops.

Volkmann's Theory of Metaplasia.—The opinions of different authors concerning the exact nature and classification of these tumors differ. Kauffmann, Nasse, Volkmann, and others regard the mixed tumors of the salivary glands as endotheliomas. They believe that the stroma may become converted into tissues of different type as the result of metaplasia, and that the columns of cells and glandlike tubules are formed by the proliferation of the endothelium lining the tissue spaces and lymphatic vessels. This view is disputed by Hinsberg, Wilms, and Ribbert. The last classifies these tumors with fibroepithelial growths and believes that they develop from displaced glandular germinal tissue, the stroma of which, because of its intimate relation to the branchial arches, is capable of forming bone and cartilage. Hinsberg holds a somewhat similar view, believing that the tumors develop from displaced islands of the parotid anlage and embryonal periosteal tissue separated from the mandible.

Mixed Tumors of the Breast.—According to Wilms, tumors of the breast, which have sometimes been described as cystosarcomas and adenosarcomas with epidermoid cysts (Grohe's cystic fibrosarcoma with epidermoidal metaplasia), sometimes as atheromas or cholesteatomas, the latter combined with cystosarcoma phyllodes (Häckel), should be classified as mixed tumors.

Clinically they are most closely related to adenomas, but they are much less common. They are found in women of middle and advanced age, occasionally in men.

They appear as nodular, well-defined tumors, which may be easily moved upon the surrounding tissues, and are covered by non-adherent skin. These tumors may grow rapidly from the beginning or after some time to attain considerable size. Finally they invade the skin and the latter ulcerates. Metastases have not been observed.

The diagnosis is not easily made. Depending upon the rapidity of growth these tumors are sometimes regarded as fibromas and adenomas, at other times as sarcomas.

Amputation of the breast is indicated to prevent recurrence.

Macroscopically the cut surfaces of these tumors vary in appearance, as sometimes they are solid, while at other times they contain spaces and round cysts. Hard and soft areas, some of which are composed of mucoid tissue, are intermingled. Microscopically these tumors differ a great deal from the adenomas, cystadenomas, and cystosarcomas which they resemble so closely clinically. Adenomatouslike tissue is found, together with epidermoid cysts with cheesy contents and cysts lined with squamous epithelium. The stroma is composed of adult and embryonal connective tissue, containing large numbers of round and spindle cells, loose mucoid tissue, cartilaginous and osteoid masses. Sometimes the blood vessels have proliferated, and the tumor simulates an angioma.

The origin of these tumors is most satisfactorily explained by Wilms. He believes that they develop from displaced ectodermal tissue, to which is also attached some mesenchyme. These fragments become enclosed within the breast tissue, and later proliferate to form glands, skin, and different types of connective tissue.

Mixed Tumors of Urogenital System.—The mixed tumors found in the urogenital system are much more malignant than those occurring in the salivary glands and breast.

They are found most frequently in the kidneys. They may develop in young children, and occasionally are of congenital origin, both kidneys being frequently involved. These tumors develop most commonly within the substance of the kidney, the renal tissue being displaced and destroyed by the new growth. The tumor, composed of large nodules and covered by the fibrous capsule of the kidney, replaces the latter organ. Frequently only a small amount of renal tissue remains at either pole. The tumor is either separated from the adjacent renal tissue by a layer of loose connective tissue, or is united to it by infiltrating masses and columns of cells. After rupture of the fibrous capsule, the tumor invades the surrounding tissue. Polypoid masses may also extend from the tumor into the pelvis of the kidney.

These tumors grow rapidly and constantly, and become very large. Finally they may fill the greater part of the abdominal cavity. They

produce lymphogenous and hæmatogenous metastases, the latter following invasion of the renal vein.

The prognosis is bad. Even the results following extirpation of the tumor and the remains of the kidney are not good, as these tumors recur rapidly. Metastases and the weakened condition of the patient contribute to the poor results of these operations.

It may be seen upon section that a number of different kinds of tissue occur in these tumors. Dense, firm, fibrous areas alternate with vascular, soft ones which resemble in structure a sarcoma.

Microscopically one finds adult and embryonal fibrous tissue with sarcomatous characteristics, mucoid tissue, cartilage, and smooth and striated muscle fibers in different stages of development. Within the stroma lie tubular glands which remind one of the canals of the primitive kidney, for their vesicular extremities, which are surrounded by connective tissue and frequently are invaginated, resemble somewhat glomeruli. The glandular content of these tumors is indicated by the number of different names, such as adenosarcoma, adenomyxosarcoma, adenomyochondrosarcoma, etc., which have been applied to them. Birch-Hirschfeld was the first to group these tumors. He called them "embryonal glandular sarcomas."

In rare cases groups and masses of cornified, flat epithelium, which contained material resembling cholesterin, have been found.

These mixed tumors must be the result of some developmental disturbance occurring during the formation of the kidney. According to Wilms, the error in development must occur very early, otherwise the number of kinds of germinal tissue displaced must correspond to the number found in the tumor. He believes that these tumors develop from germinal mesoderm separated and displaced from the vicinity of the primitive kidney.

Mixed Tumors of the Vagina.—Mixed tumors of the vagina appear in small children as grapelike growths, which, like those developing upon the cervix of middle-aged people, grow rapidly, fill the vagina, protrude between the labia, and later infiltrate the pelvic connective tissues. Both forms recur rapidly, ulcerate, and become infected early, and cause death in a few years. Metastases rarely develop.

The origin of these tumors, composed of different forms of sarcomatous tissue, together with muscle fibers, cartilage, mucoid tissue, and fat, may be most satisfactorily explained by Wilms's theory, according to which they develop from an undifferentiated, germinal, mesodermal tissue which is displaced during early development.

A similar histological picture is presented by the rare mixed tumors occurring in the urinary bladder, which appear about the trigone. According to Wilms these also develop from displaced mesodermal tis-

sue. This investigator ascribes the same origin to mixed tumors developing in the lower pole of the testicle and along the vas deferens of young children, which, depending upon the character of the tissues composing them, are usually called rhabdomyomas or rhabdomyosarcomas.

Ribbert has suggested that mixed tumors of the urogenital system, which are apt to occur at definite points, develop from separated germinal cells which migrate from the region of the kidney through the Müllerian and Wolffian ducts to the uterus, vagina, and urinary bladder, and perhaps even gain access to the vas deferens.

LITERATURE.—*Birch-Hirschfeld.* Sarkomatöse Drüsengeschwulst der Niere im Kindesalter (embryonales Adenosarkom). Zieglers Beitr. z. path. Anat., Bd. 24, 1898, p. 343.—*Hinsberg.* Beiträge zur Entwicklungsgeschichte und Natur der Mundspeicheldrüsengeschwülste. Deutsche Zeitschr. f. Chir., Bd. 51, 1899, p. 281.—*Hüsler.* Beitr. z. Lehre von d. Harnblasengeschwülsten im Kindesalter. Jahrb. f. Kinderheilk., Bd. 62, 1905, Part 2.—*Kaufmann.* Das Parotissarkom. Arch. f. klin. Chir., Bd. 26, 1881, p. 672.—*Küttner.* Die Geschwülste der Submaxillarspeicheldrüse. Beitr. z. klin. Chir., Bd. 16, 1896, p. 181.—*Nasse.* Die Geschwülste der Speicheldrüsen, etc. Arch. f. klin. Chir., Bd. 44, 1892, p. 233.—*Volkmann.* Ueber endotheliale Geschwülste, zugleich ein Beitrag zu den Speicheldrüsen- und Gaumentumoren. Deutsche Zeitschr. f. Chir., Bd. 51, 1895, p. 1.—*M. Wilms.* Die Mischgeschwülste, I–III. Berlin und Leipzig, 1899, 1900, 1902.—Weitere Literatur siehe bei *Borst.* Die Lehre von den Geschwülsten. Wiesbaden, 1902, II, p. 979.

CHAPTER II

TERATOID TUMORS

(a) COMPLICATED DERMOID CYSTS OF THE OVARIES AND TESTICLES

(Wilms's Cystic Embryomas)

THE most striking characteristic of these growths, which are usually benign, is their similarity to simple dermoid cysts, which consist only of follicles of skin provided with hair and glands and have cheesy contents. The presence, however, of large amounts of hair, of teeth, and particles of bone indicates the complicated structure of these tumors, and for this reason they are differentiated from the simple dermoid cysts.

Common Occurrence in Ovaries.—They are found most frequently in the ovary, forming nine per cent of all ovarian tumors. These tumors are found more rarely in children than in adults, but apparently all of them are the result of anomalies in development. They develop as single or multiple growths in one or both ovaries, and may be situated within

the latter organs as well as upon their surfaces. When situated upon the surface of the ovaries they are frequently pedunculated. When the pedicle is broken, they lie free in the lower, occasionally in the upper abdomen (e. g., in the lesser peritoneal cavity). The cysts which lie at a distance from the ovaries may have developed from accessory ovaries.

These cysts usually grow slowly, but may become as large as a man's head. Then the ovary is partially or completely destroyed. Occasionally the cyst wall ruptures, and then viable germinal tissue from which other small cysts develop is transplanted upon the peritoneum. Carcinomas, usually of the squamous-cell variety, may develop from the walls of these cysts.

Symptoms—Torsion of Pedicle, etc.—The first symptoms are usually produced by pressure upon neighboring organs after the cysts attain considerable size, or by secondary disturbances which may be followed by serious consequences. Pedunculated cysts are frequently deprived of nutrition by torsion of their pedicles. When torsion occurs the cyst becomes necrotic, contracts adhesions with neighboring structures, or may rupture in the bladder or intestines.

Frequently cysts after torsion of the pedicle are invaded by intestinal bacteria, and then suppurate or undergo putrefactive changes. A progressive fatal peritonitis may follow infection of one of these cysts.

It is difficult to make a differential diagnosis between dermoid cysts and other tumors which occur in the ovary and tissues adjacent to it.

Treatment.—The treatment consists of complete removal, together with the remnants of the ovary involved.

Complicated Dermoids of the Testicles.—The complicated dermoid cysts of the testicle are much rarer. They are more rare in adults than in small children, in whom they are frequently congenital. Occasionally they develop in undescended testicles. They occur as single growths and are found only within the testicles, not occurring upon the surface, and usually develop from the substance of the organ, only small remnants of the latter remaining upon the surface of the tumor. Tumors situated without the testicle—for example, in the scrotum—are rare.

They grow slowly, the patient's attention usually first being attracted to the growth by its size, and never give rise to the severe symptoms which are sometimes associated with similar growths of the ovaries. They may be differentiated from other slowly growing tumors of the testicles by their doughy consistency.

Castration is indicated in the treatment of these tumors.

Macroscopic Appearance and Histology.—According to the investigations of Wilms, the complicated dermoid cysts of the testicles and ovary are peculiar in that they contain a rudimentary embryonal anlage, and therefore he has called these growths *cystic embryomas.*

The inner surface of the firm cyst wall, which is not of the same thickness throughout, contains a number of tumorlike projections. Sometimes these projections resemble villi, sometimes nodular thickenings, while at other times they appear as septa between cavities within the cyst. These projections are covered with skin, which is frequently covered with masses of hair, and often contain teeth connected with pieces of bone which extend into the deeper parts of the cyst wall. Cysts of this character contain derivatives of the three germinal layers. These derivatives resemble the structures of the cephalic portion of an embryo, but do not have the irregular arrangement found in teratoid mixed tumors (Wilms). Subcutaneous fat, cartilage, bone, muscle fibers, and even brain tissue with corpora amylacea may be found beneath the skin lining such a cyst. In some parts of the cysts the skin becomes continuous with mucous membrane covered with squamous epithelium, and the latter with cylindrical or ciliated epithelium lining a canal, the character and arrangement of the epithelium being similar to that found in the mouth cavity of the embryo. Some of the cysts, the walls of which contain rings of cartilage and smooth muscle fibers, resemble in structure the respiratory passages, while others, lined with goblet cells and filled with mucus, the walls of which contain smooth muscle fibers, resemble histologically the intestines. Widely different types of tissues may be found which have often been regarded as rudiments of an eye, of the thyroid gland, trachea, mammary gland, etc. The greater part of the walls of these cysts is lined with skin, the remaining part is lined with cylindrical epithelium or covered with granulation tissue.

Naturally the different tissues do not reach the same degree of development in the different tumors. The embryonal tissues and organs which develop earliest predominate, viz., the ectoderm and the tissues and organs of the cephalic region. The development of the other tissues is prevented by the rapid growth of those which differentiate early (Wilms).

Theories as to Origin.—Different theories have been advanced to explain the development of these complicated dermoid cysts (embryomas) in the testicles and ovaries. It has been suggested that they are the result of abnormal proliferative changes in the spermatozoa or ova, which occur without fertilization, the process having been called *parthenogenesis.*

The possibility of the parthenogenetic origin of cysts of this character has been disputed by Bonnet. He believes that these tumors develop from blastomeres which have not gone on to full differentiation or from fertilized polar globules. Marchand and Wilms believe that the tumors occurring in the testicle and ovary may develop from excessive, unused blastomeres. They do not accept Bonnet's view that they may develop

from polar globules, as cases have been observed in which five embryomas have been found in an ovary, while in the normal human ovum usually only two, never more than three, polar globules are formed (Wilms).

According to Wilms these tumors develop from germinal tissue containing the three layers which is displaced during development. This theory explains most satisfactorily not only the development of the complicated dermoid cysts found in the ovaries and testicles, but also that of the teratoid mixed tumors occurring in these organs. Teratoid tumors and teratomas are supposed by some to develop from unused, displaced blastomeres, by others from fertilized polar globules. The genetic relationship between these tumors and dermoids of the ovary is demonstrated by the occurrence within the abdominal cavity of tumors, which are morphologically similar to dermoids of the ovary, but have no connection with the latter organ. It has not been satisfactorily explained why the displaced germinal tissue produces such different macroscopic and microscopic pictures when it proliferates.

(b) TERATOID MIXED TUMORS

(*Embryoid Tumors, Wilms*)

The tumors placed in this group differ from the complicated dermoid cysts in that they are solid or polycystic; from teratomas in the absence of any highly developed rudiments. They are formed as a result of the irregular proliferation of derivatives of the three germinal layers (therefore, tridermoma, Wilms). They have a much more varied structure than the simple mixed tumors.

These tumors may develop in any part of the body in which teratomas occur, but are found most frequently in the testicles and ovaries.

Teratoid Mixed Tumors of Testicles.—Teratoid mixed tumors of the testicles develop most commonly between the twentieth and fortieth years of life. They grow slowly to form large nodular tumors of irregular size and varying consistency. The changes produced in the surrounding tissues by these tumors are very similar to those produced by complicated dermoids. They become malignant with relative frequency. These tumors may assume a sarcomatous, more rarely an adenocarcinomatous structure, and may rupture through the capsule formed by the tunica albuginea, may infiltrate the surrounding tissues and form numerous metastases, which often correspond histologically to the malignant part of the tumor only. Frequently, however, the metastases contain the derivatives of the three germinal layers. Death soon follows the formation of metastases.

The varying consistency of the slowly growing tumors is the most important diagnostic point. When these tumors assume malignant characteristics they cannot be differentiated from sarcomas and carcinomas of the testicle.

Macroscopical Appearance and Histology.—Depending upon the character of the tissue which predominates, these tumors have been called chondromas, cystosarcomas, chondroadenomas, adenocystomas, adenomyosarcomas, cystocarcinomas, etc., of the testicle. Wilms has shown that almost all of these tumors contain derivatives of the three germinal layers, but when the derivatives of the entoderm and mesoderm predominate those of the ectoderm may be wanting. The different tissues found in these tumors have a very irregular arrangement and structure. The stroma may contain embryonal and adult forms of fibrous tissues, myxomatous tissue and fat, cartilage and bone, smooth and striated muscle fibers, and remnants of the peripheral and central nervous system. Scattered throughout the stroma may be found glandular structures varying in form and arrangement, spaces and cysts lined with different forms of epithelium, the contents of which differ. Frequently the mucous membrane found in these tumors resembles, histologically, that of the mouth and pharynx or intestinal tract.

Teratoid Mixed Tumors of the Ovary.—Teratoid mixed tumors of the ovary, like complicated dermoid cysts, develop within the organ, upon its surface, or in the surrounding tissues. They develop most frequently during puberty, and grow slowly, unless they undergo malignant changes.

Upon section these tumors have a mottled appearance, and derivatives of the three germinal layers may be found. Occasionally these derivatives, especially those of a cephalic region, are fairly well developed, being similar to the structures already described in complicated dermoid cysts.

Occurrence in Other Organs.—Teratoid mixed tumors occur in the buccal, nasal, and pharyngeal cavities as hairy polyps (Arnold). They are also found within the skull, being situated within the ventricles or at the base of the brain, appearing as tumors of the hypophysis. They may be found within the chest (within the mediastinum or pericardium) and in the abdominal cavity (in the transverse mesocolon, the lesser peritoneal cavity, in the mesentery or retroperitoneal tissues upon the left side, close to the spinal column). Tumors occurring in the abdomen develop from parts of fœtal abdominal organs displaced during the rotation of the intestines and the changes occurring in the fœtal peritoneum, which terminate in its fusion with the posterior abdominal wall. Tumors of this class are also found in front of and behind the coccyx and sacrum; in short, wherever teratomas occur. It

should be mentioned that B. Fischer has found one of these tumors in the muscles of the calf of a male patient.

Removal by operation, which may be very difficult, is always indicated.

CHAPTER III

TERATOMAS

The tumors of this group, which are rare, form a transition between the teratoid mixed tumors and the double malformations, such as a *fœtus in fœtu*. They are therefore on the border line between new growths and malformations. Some of the double malformations may assume tumorlike growths.

Teratomas are always congenital, that is, the tumor is present at the time of birth or the patient is born with the essential tumor matrix.

Position.—These tumors either lie near the surface of the body—for example, teratomas of the mouth which are attached to the base of the skull, teratomas of the neck and coccygeal region—or within the body cavities (pleura and peritoneum).

Size and Form.—Teratomas may be of considerable size at birth, or if the child survives may gradually become of considerable size during subsequent growth.

Shape and Consistency.—These tumors vary in shape, their surfaces usually being nodular and irregular. As they are composed of cystic and solid areas, the consistency of various tumors differs. Frequently a dense connective-tissue capsule surrounds such a tumor, separating it from the surrounding tissues. Large vessels enter the capsule, usually at the point at which the tumor first commenced to develop. A teratoma of the abdominal cavity may have an amnionlike membrane surrounding it (Fig. 444). When one of these tumors develops near the surface of the body the skin may form a capsule.

Differences Between Teratomas and Teratoid Mixed Tumors.—Teratomas differ from teratoid mixed tumors in containing highly developed fœtal structures. It is not always possible, however, to make a sharp distinction between these two classes of tumors. Teratomas contain incompletely developed parts of the skeleton, such as the skull, mandible, bones of the extremities, entire extremities or parts of the same with joints, fingers, or toes in different stages of development; intestinal loops with a mesentery; cysts resembling, histologically, stomach, lung, thyroid, kidney, and pancreas tissue; pieces of the brain with convolutions and ventricles; anlage of the bronchi, eyes, nerves, etc.

The parts covered with skin project into a dermoidlike cavity or are surrounded by amnion. These different kinds of tissue are held together by tumorlike masses which correspond to the different germinal layers from which they develop.

One gains the impression, when such a tumor is examined, that the growth of the cells of one of the germinal layers has been suppressed or that the cells have been partially destroyed, and that the layers

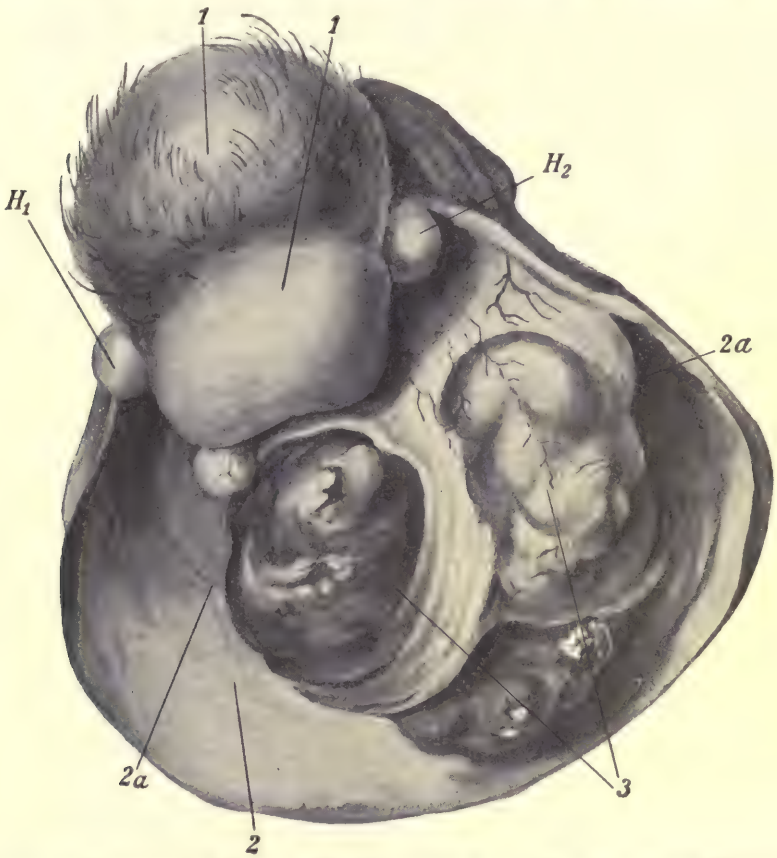


FIG. 444.—A TERATOMA THE SIZE OF A FIST REMOVED FROM A GIRL BABY SEVEN WEEKS OLD BY OPERATION. The tumor lay in the foramen of Winslow, just beneath the liver and was adherent to the hepato-duodenal ligament, from which its nutrient vessels were derived. (Lexer.) *1*, The pedunculated sac of skin contains an anlage of the head, consisting of brain cavities, connective-tissue skull, well-developed scalp, and anlage of teeth which extend into the pedicle and squamous cell epithelium, which resembles that of the mouth. *2*, Capsule of the tumor open. *2a*, Point of attachment of tumor. The tumor also contains a cyst situated close to the anlage of the head which is provided with ciliated epithelium and goblet cells, mucous glands, smooth muscle fibers and hyaline cartilage (anlage of the respiratory passages). The principal part of the nodular tumor is a teratoid (polycystic) mixed tumor. H_1, H_2, Nodules of skin attached to the head anlage.

which possessed sufficient growth energy have proliferated to form imperfect organs, which, however, have no definite morphological or functional relations with each other.

Diagnosis.—The correct anatomical diagnosis depends upon the position of the tumor. The nature of a superficial tumor occurring in areas in which tumors of this character are common is recognized earlier than that of a similar tumor situated in the pleural or peritoneal cavities. Of course tumors of the pleural cavities are inaccessible. If extremities or teeth can be demonstrated in tumors of the abdomen by palpation or by Röntgen-ray examination, a diagnosis can be made with some degree of certainty. In some of the cases, however, the parts are so rudimentary that it is difficult to interpret the findings.

Teratomas have frequently been removed by operative procedures, but only a few of the cases have been successful.

Origin of Teratomas.—A teratoma never springs from a postnatal matrix. Many of the tumors occurring about the cephalic and caudal extremities develop from germinal tissue which has been displaced during fusion of the germinal plates, and from embryonal structures which normally undergo involution, such as the neurenteric canal, the postanal gut, the medullary tube, and the caudal vertebræ.

Monogerminal and Bigerminal Tumors.—Tumors developing from displaced rests or from structures which normally undergo involution are called *monogerminal* tumors, while those which resemble fœtal inclusions are called *bigerminal* tumors (bigerminal teratomas or fœtal inclusions, parasitic implantations, parasites, *fœtus in fœtu*).

It is not always easy to differentiate between these two groups. The differentiation is usually based upon the following principle: If the tissues or organs found in the tumor resemble those normally found in the area where the tumor is situated, the latter is regarded as a monogerminal tumor, while if the tissue found in the tumor is foreign to the area in which the former occurs it is called a bigerminal tumor.

LITERATURE.—*Arnold.* Behaarte Polypen der Rachen-Mundhöhle. Virchows Archiv, Bd. 111, 1888, p. 176.—*Aschoff.* Zysten. Ergebn. d. allg. Path., II, 1897, p. 456.—*Borst.* Die angeborenen Geschwülste der Sakralregion. Zentralbl. f. allg. Path., Bd. 9, 1898, p. 459.—*Engelmann.* Beiträge zur Kenntnis der Sakraltumoren. Arch. f. klin. Chir., Bd. 72, 1904, p. 942.—*B. Fischer.* Ueber ein Embryom der Wade. Münch. med. Wochenschr., 1905, p. 1569.—*Kirmisson.* Chirurgische Krankheiten angeborenen Ursprunges. Stuttgart, Enke, 1899.—*Kleinwächter.* Ueber operierte Kreuzbeinparasiten, etc. Zeitschr. f. Heilkunde, Bd. 9, p. 1.—*Lexer.* Ueber teratoide Geschwülste in der Bauchhöhle und deren Operation. Arch. f. klin. Chir., Bd. 61, 1900, p. 648;—Operation einer fötalen Inklusion in der Bauchhöhle. Arch. f. klin. Chir., Bd. 62, 1900, p. 351.—*Linser.* Ueber Sakraltumoren und eine seltene fötale Inklusion. Beitr. z. klin. Chir., Bd. 29, 1901, p. 388.—*Marchand.* Sakraltumoren. Eulenburgs Realenzyklopädie, Bd. 25, 1899.—*Achilles Müller.* Zur Kenntnis der Hodenembryome.

Arch. f. klin. Chir., Bd. 76, 1905, p. 661.—*Nasse.* Beiträge zur Genese der sacrococcygealen Teratome. Arch. f. klin. Chir., Bd. 45, 1893, p. 985.—*Otto.* Ueber einen kongenitalen behaarten Rachenpolypen. Virchows Archiv, Bd. 115, 1889, p. 242.—*Pupovac.* Ein Fall von Teratoma colli mit Veränderungen in den regionären Lymphdrüsen. Arch. f. klin. Chir., Bd. 53, 1896, p. 59.—*Saxer.* Ein zum grössten Teil aus Derivaten der Medullarplatte bestehendes grosses Teratom im 3. Ventrikel eines 7wöchentlichen Kindes. Zieglers Beitr. z. path. Anat., Bd. 20, 1896, p. 399.—*Wetzel.* Zur Kasuistik der Teratome des Halses. I.-D., Giessen, 1895.—*Wilms.* Dermoidzysten und Teratome. Deutsch. Arch. f. klin. Med., Bd. 55, 1895, p. 289;—Ueber die soliden Teratome des Ovarium. Zieglers Beitr. z. path. Anat., Bd. 19, 1895, p. 367;—Die teratoiden Geschwülste des Hodens. *Ibid.*, Bd. 19, 1896, p. 233;—Embryome und embryoide Tumoren des Hodens. Deutsche Zeitschr. f. Chir., Bd. 49, 1898, p. 1;—Multiple Embryome des Ovarium. Monatsschr. f. Geburtsh., Bd. 9, 1899, p. 585.—Weitere Literatur siehe bei *Borst.* Die Lehre von den Geschwülsten. Wiesbaden, 1902, II, pp. 980–982.

PART VII

CYSTS, NOT INCLUDING CYSTIC TUMORS

Definition—Unilocular and Multilocular Cysts.—A cyst is a circumscribed cavity, the contents of which may be thin, thick, or atheromatous, separated from the surrounding tissues by a connective-tissue membrane or by tissue of complex structure. Cysts with but a single cavity are called *unilocular*, while cysts with many cavities are called *multilocular*. Cysts, as they develop, tend to become spherical, but the form is modified by the resistance offered by surrounding structures, such as fascia and bone, by adhesions which the wall of the cyst contracts, and by the form of the original cavity.

There are a number of varieties of cysts which have no relation whatever to true tumors, besides the cysts which develop as the result of peculiarities of growth in true tumors (embryonal cysts, cystadenomas) and as the result of softening and liquefaction in solid tumors.

Four varieties of cysts, which include the false as well as the true, may be differentiated. A true cyst has either an epithelial or endothelial lining, while the connective-tissue capsule of the false cyst has no lining at all.

(1) VIRCHOW'S EXUDATION OR EXTRAVASATION CYSTS

Cysts of this character are formed when an exudate or blood is poured out into a preëxisting cavity, or one formed as the result of some pathological process. A hydrocele of the tunica vaginalis testis or of the spermatic cord is the best example of an exudation cyst. The serous exudate formed during a chronic inflammation fills the remains of the processus vaginalis peritonei. In hygromas—cysts of the bursæ and tendon sheaths—the serous or serohæmorrhagic exudate formed during the chronic inflammation is likewise poured out into a preëxisting cavity. An ascites, a hydrops of a joint, or a hydrocephalus might be spoken of as a cyst, but the anatomical relations, which are not the same as in cysts, do not permit of it. On the other hand, an empty hernial sac or a meningocele may be shut off from the cavity with which it formerly communicated; the fluid secreted by the endothelial lining can then no longer escape, and the hernial and meningeal sac becomes transformed in a cyst.

Hæmatocele.—A hæmatocele follows the exudation of blood into the tunica vaginalis testis. The contents of a hæmatocele consist partly of fluid, partly of friable, coagulated blood. The tunica vaginalis becomes thickened by deposits of fibrin and as the result of connective-tissue proliferation. If blood is poured out between the ends of a muscle which has been lacerated or contused without separation of the overlying skin, a *traumatic blood cyst* is formed. The blood and necrotic tissue, which later becomes liquefied, are surrounded by a connective-tissue capsule, the result of a reactive inflammation.

Pneumatocele.—The rare circumscribed collections of air between the bones of the skull and the overlying periosteum, which are known as pneumatoceles, may also be classified with this variety of cysts. They occur on the forehead and in the occipital region just behind the ear, and communicate with the frontal sinus and the air cells of the mastoid, respectively, by small holes in the bones. The clear tympanitic note, the increase in the size of the swelling during expiration when the nose and mouth are closed, the decrease in size of the swelling when pressure is made, and the position of the swelling are the most important characteristics. A pneumatocele may be cured by simple incision, as the granulation tissue which forms following the use of a tampon may close the opening in the bone. If the swelling returns, an osteoplastic operation may be required (von Bergmann).

(2) LIQUEFACTION CYSTS

The second variety of cysts comprises those which result from the softening and liquefaction of tissue. Softening and liquefaction follow nutritional changes in the tissues. The thin or colloidlike masses produced by the softening and liquefaction of the dead tissue are surrounded by a connective-tissue capsule which varies in thickness and degree of development in different cases. Liquefaction cysts frequently develop in tumors.

Cysts developing in the brain secondary to ischæmic softening and the colloid cysts of capsular ligaments and joints, known as ganglia, which are closely related to hygromas developing in bursæ, belong to this class. Abscesses, the contents of which consist partly of liquefied necrotic tissue and partly of inflammatory exudate, are closely related to this variety of cysts.

(3) RETENTION CYSTS

If the duct or canal of a gland or cavity which secretes or contains a fluid becomes closed and the secretion or fluid continues to form, the

gland or cavity becomes dilated to form a cyst (Virchow's dilatation and retention cysts). The tissues composing the wall of the gland or cavity proliferate as the result of the irritation produced by the accumulation of fluid, and the cyst enlarges until further growth is prevented by physiological or anatomical conditions.

Cysts of this variety are found in cavities, glands, and blood vessels.

(a) Hydrops Vesicæ Felleæ, Pyosalpinx, etc.—Hydrops of the gall-bladder is the best and most common example of cystic changes in a hollow organ following the occlusion of its duct. If the lumen of the cystic duct is closed by a gallstone or occluded by cicatricial contraction of its walls, the walls of the gall-bladder become distended by the mucus which collects and the gall-bladder becomes transformed into a structure resembling in form a cucumber (hydrops vesicæ felleæ, Fig. 445). Similar changes may occur in the vermiform appendix following cicatricial stenosis of its lumen. The appendix then becomes transformed into a cyst, resembling a pear in shape, which is filled with masses of mucus (hydrops processus vermiformis). These changes occur frequently in the Fallopian tubes following obliteration of both the abdominal and the uterine ends. The cysts thus formed are called, depending upon the

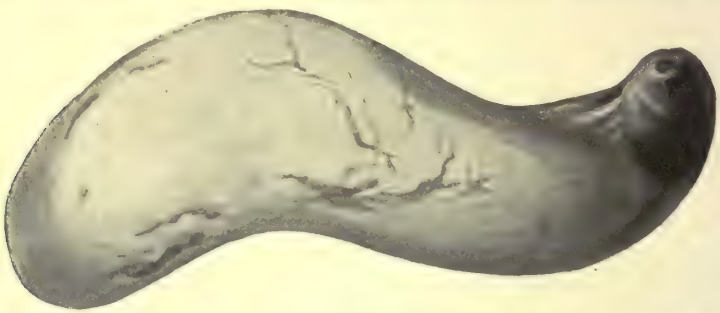


FIG. 445.—HYDROPS OF THE GALL-BLADDER. (One half natural size.) A gallstone may be seen showing through the beginning of the cystic duct.

character of their contents, hydrops tubarum, pyosalpinx, and hæmatosalpinx. Hydronephrosis is another example. In hydronephrosis the escape of urine is permanently or intermittently, completely or partially prevented by some congenital anomaly, torsion, or cicatricial stenosis of the ureter or by the incarceration of a stone. The urine gradually collects in the pelvis of the kidney, resulting in a marked dilatation of the latter, and a gradual destruction of the parenchyma of the kidney occurs, so that in advanced cases only a few remnants of kidney tissue remain.

(b) Retention cysts of glands develop either in the ducts or in the bodies of the glands.

1. *Sebaceous Cyst or Atheroma.*—An atheroma is a retention cyst of a sebaceous gland, the cyst developing either in the duct or body of the gland. A comedo, which appears as a black point in the skin, and may be expressed together with a wormlike mass of secretion, indicates the beginning of retention which may eventually end in cyst formation.

Mode of Formation, Lining and Contents of Cysts.—The excretory duct of the sebaceous gland may be blocked as the result of the formation of the crusts, of uncleanliness, or of inflammation. If the accumulation of secretion is limited to the excretory duct, a small cyst filled with fatty contents, which never becomes larger than a hemp seed, forms. According to Chiari, if the body of the gland is involved the orifice of the duct dilates first, the body of the gland later. A cyst then develops which lies in the corium and is attached to the upper layers of the skin by the outer occluded parts of the duct of the gland. The cyst may, however, gradually enlarge and finally extend into the subcutaneous tissues. The cyst wall, which is but loosely attached to the surrounding tissues, consists of a thin layer of fibrous connective tissue, while its inner surface is lined by a number of layers of flattened epithelial cells with the stratum Malpighii, but containing no papillæ. Remnants of the sebaceous gland and hair follicles may also be found in the wall of the cyst. These cysts contain a yellowish white or grayish, fatty, salvelike mass, which has been compared to cooked barley. Frequently the mass is foul-smelling and mixed with pus. The earlier the remnants of the sebaceous gland disappear from the cyst the more cornified the contents of the cyst, which may be recognized by the dry characteristics and the stratification.

Fig. 446.—Atheroma of the Ear.

The smallest atheromas, which appear as round, hard, white nodules in the skin, may be easily recognized. These may gradually enlarge to become as large as a cherry or walnut, occasionally as large as a fist, and are covered by and attached to tense, white or, because of stasis in the small veins, bluish discolored skin.

Regressive Changes.—Calcification of the cyst wall, *suppurative* and *putrefactive* inflammation, which may follow attempts to evacuate the contents of cysts by digital pressure or massage applied to thin skin, are some of the secondary changes which should be mentioned. Frequently the hair follicle becomes so dilated following an inflammation or attempt at expression of the contents of the cyst that a wide defect

forms, in the base of which the dried and discolored atheromatous masses may be seen. A carcinoma occasionally develops from a sebaceous cyst, but the diagnosis cannot be made until the growth has ruptured through the cyst wall and invaded the skin. When a cyst enlarges rapidly and becomes irregular in form, suspicion of malignancy should be aroused.

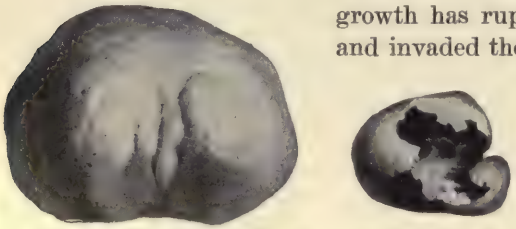


FIG. 447.—ENUCLEATED SEBACEOUS CYSTS.

Most Common Sites for Development. — Sebaceous cysts develop most frequently in the scalp. They are more common in women than in men. The skin of the face in the region of the ear, of the cheek, eyelids, neck, back, and external genitalia are involved most commonly after the scalp, in the order of frequency as given here. Multiple atheromas of the scalp and skin of the back are common (Fig. 448).

FIG. 448.—MULTIPLE ATHEROMAS (FOURTEEN) OF THE SCALP.

Age. — Atheromas have not been observed in the young (never before the fifteenth year—Chiari). They occur very frequently in the old.

Diagnosis. — The diagnosis is easily made, as the cysts are round and sharply defined, have smooth surfaces, are intimately related to the cutis covering them, move freely with the skin upon the subjacent tissue, occur in definite parts of the body, and have a slow, painless growth, unless inflamed or undergoing malignant changes. Small cysts have a hard, large ones a doughy or fluctuating consistency. Cysts lying close together may apparently communicate with each other (Fig. 448).

The diagnosis is difficult only when no connection can be demon-

strated between the cyst and the skin, and the latter can be entirely raised from the surface of the former. If this is the case, the cyst may be a dermoid, a subcutaneous atheroma, the duct of which has been constricted off, or an epidermoid. A positive clinical diagnosis cannot be made in these cases, and a microscopic examination is necessary. Papillæ are never found in the epithelial lining of an atheroma, while they are usually present in epidermoids.

Indications for Removal—Technic.—The removal of sebaceous cysts may be indicated for cosmetic reasons, because of pain following inflammation or the beginning of malignant changes. The entire cyst wall must be removed in order to prevent recurrences. [In removing a cyst, the latter should be transfixed, the cheesy contents squeezed out, and the cyst wall removed by grasping it and pulling it away with artery or tissue forceps. In removing a cyst wall, the inspissated, stratified contents of the cyst should not be mistaken for the epithelial lining.]

In some situations the cyst wall will have to be dissected out, but even in these cases it is better to transfix the cyst and deal with the wall from below upward. If the cyst wall has been partially destroyed by suppuration, it may be necessary to remove the remnants of the epithelial lining with a sharp spoon.

2. *Mucous Cysts.*—Retention cysts not infrequently develop within the mucous glands. Small multiple cysts or single large growths may develop after chronic inflammation of the mucous membrane, associated with atrophy, and in polypoid growths of the nose and accessory sinuses. Large solitary cysts are common in the mouth cavity, occurring frequently upon the inner surfaces of the lips, especially of the lower lips of children and adults; in the cheeks and upon the posterior part of the lower surface and the margins of the tongue. These cysts produce round, painless, circumscribed projections, which grow slowly and rarely become larger than a bean. The mucous contents of the cysts shine through the thinned mucous membrane which covers them. Mucous cysts have thin walls which are usually lined by but a few epithelial remnants, as the greater number of the epithelial cells are cast off into the cavity of the cysts and degenerate.

Symptoms develop only when the mucous membrane covering these cysts is injured during mastication.

The nature of the sharply delimited cyst, which appears as a vesicle, is usually easily recognized. A mucous cyst may resemble a cystic lymphangioma, but the tissues surrounding the latter usually contain cavernous tissue and are thickened, while the area surrounding the former is normal. The size of the cyst cannot be reduced by pressure. The contents of a mucous cyst cannot be expressed by pressure unless

ruptured, while those of a cavernous hæmangioma or of an aneurysm of one of the labial arteries can.

Removal.—Mucous cysts can be removed easily. The cyst wall is, however, usually so thin that enucleation is impossible, and therefore it is recommended that the external wall of the cyst, together with mucous membrane covering it, be cut away with curved scissors and that the remainder of the cyst wall be destroyed with the actual cautery.

3. *Cysts of Salivary Glands—Ranulæ.*—Retention cysts of the salivary glands may develop in the excretory ducts as well as in the glands proper, and, therefore, cysts of the ducts are differentiated from cysts of the glands.

Cysts develop most frequently in the ducts of the submaxillary and parotid glands after injuries, inflammatory processes, and the impaction of salivary calculi. A long oval swelling develops, which gradually enlarges, associated with a swelling of the corresponding glands due to the accumulation of saliva. The mucous membrane covering such a cyst may be easily injured, and the first difficulties are usually experienced when the cyst becomes inflamed or becomes large enough to project prominently into the floor of the mouth. Frequently these cysts rupture into the floor of the mouth, undergoing then spontaneous cure, as an internal salivary fistula develops. If a cyst of the parotid duct ruptures upon the cheek, and an external salivary fistula is formed, an operation will be required to close it. Destruction of the salivary gland following obstruction of its duct may end in spontaneous cure.

The long form and the position of the cysts of the excretory ducts of the salivary glands are characteristic, and enable one to differentiate them with certainty from other forms of cysts.

Incision of the wall of the cyst, producing an internal salivary fistula, is the treatment indicated in these cases.

Cysts of the glands proper are the sequelæ of chronic inflammatory processes, for the proliferating connective tissues compress the smaller ducts and occlude their lumina. Usually many ducts are involved simultaneously, and as the result of pressure atrophy of the tissues separating the smaller cysts a number of the latter may fuse to form one large one. The cells lining these large cysts gradually become flattened by pressure, while the connective tissues surrounding them proliferate to form a thin capsule, which is attached to the adjacent tissues. The formation of the larger cysts by the coalescence of smaller ones is indicated by projections, resembling septa, which are found within the walls of the former.

The majority of these cysts are found within the sublingual gland. They form the greater part of those cysts in the floor of the mouth which are classified as ranulæ. Cysts of the sublingual gland usually

first appear at the side of the frenulum. As they enlarge they may produce a marked swelling upon both sides of it, which projects prominently into the floor of the mouth. The cysts are usually covered by a thin, tense mucous membrane, while the cysts of the submaxillary and parotid glands, which never become larger than a walnut, lie concealed beneath normal skin, to which they are not attached. All these cysts grow slowly and do not give rise to any marked symptoms until they have reached considerable size. A clear fluid containing threads of mucus is found in these cysts.

Cysts of the sublingual gland are easily recognized. Lipomas and dermoid cysts of the floor of the mouth, which should be considered in a differential diagnosis, do not have the bluish, transparent appearance so characteristic of ranulæ. Retention cysts of the ducts of submaxillary glands have a long, oval form corresponding to the position of the ducts, which is quite characteristic. It is more difficult to recognize cysts of the parotid and submaxillary glands, and frequently a diagnosis cannot be made without an exploratory puncture. The cysts may be situated in the deeper parts of the enlarged glands, and it may be very difficult in these cases to differentiate retention cysts from true tumors.

Complete removal of the cyst or destruction of the entire wall is the only treatment which is at all successful. Incision alone is never successful, as the cyst recurs as soon as the incision heals. Injection of tincture of iodin may be tried in the treatment of cysts of the parotid gland. If the cysts are situated in the submaxillary or sublingual glands, complete extirpation of the cyst with the corresponding gland is to be recommended.

Cysts of the Glandular Organs.—Many of the different forms of retention cysts which occur within the different glands are of surgical importance.

(*i*) *Retention Cysts of the Breast.*—Two forms of retention cysts are found in the female breast. The retention cyst containing milk, called a *galactocele*, follows the occlusion of the smaller or larger milk ducts of the functionating breast by inflammatory processes or cicatricial changes. In the other form *multiple cysts* develop. These are found most frequently in old people, and usually develop in both breasts. The multiple cysts vary in size. The majority, however, are small and may be easily recognized by their dark brown or green color, when the surface of the breast is exposed or the gland is incised. According to König, these multiple cysts follow the occlusion of the finer ducts by a chronic inflammatory process which also stimulates the epithelium to the production of a serous or colloid material. The cicatricial changes ending in obliteration of the ducts also occur in the involuting breast. The origin of these cysts has not been satisfactorily explained, and many—for

example, Schimmelbusch—believe that the proliferation of the epithelium lining the acini is the primary, the cystic dilatation the secondary change. He therefore thinks that multiple cysts should be regarded as cystadenomas.

(*ii*) *Retention Cysts of the Testicle.*—The retention cysts, which not infrequently develop within the testicle, are known as spermatoceles. They are usually found within the globus major of the epididymis and in the rete testis. As they develop they displace the testicle downward. They are regarded as retention cysts of the vasa efferentia or of the aberrant ducts, one extremity of which ends blindly, the other communicates with the ducts of the testicle or epididymis. These cysts grow slowly without causing symptoms and vary in size from a bean to a man's fist. Frequently spermatozoa can be demonstrated in the milky fluid which they contain. They may be lined with cylindrical cells with or without cilia or with flat epithelium. These cysts are usually secondary to obliteration of the ducts by inflammatory processes following gonorrhea.

(*iii*) *Retention Cysts of Pancreas and Liver.*—Cysts of the pancreas, following occlusion of the larger ducts or their branches, or of the interlobular ducts by interstitial inflammatory processes, are rare. Some of the cysts found in the liver, follow obliteration of the finer bile ducts by cicatricial tissue. [The majority of the cysts found within the pancreas and liver are new growths and should be classified with the cystadenomas.]

(*iv*) *Retention Cysts of Ductless Glands.*—Retention cysts may develop in organs which have no excretory ducts. Cysts of the ovary, *hydrops follicularis*, are common. These may occur as multiple cysts in the ovary developing from follicles which have not ruptured. They may become as large as a fist. Not infrequently multiple cysts develop in the thyroid gland from the fusion of dilated follicles containing colloid.

The diagnosis and treatment of cysts of the different glands belong to special surgery.

(c) Lymphatic, Blood, and Chylous Cysts.—It is not to be doubted that retention cysts occur in lymphatic and blood vessels, but it is difficult to recognize the causes of the different forms. Blood, lymphatic, and chylous cysts are the ones which follow the extravasation of blood and lymph or occlusion of the vessels by disease.

Frequently blood cysts follow the extravasation and encapsulation of blood in the different tissues. Blood cysts following the extravasation of blood into the peritoneum or loose retroperitoneal tissues after subcutaneous injuries of the abdomen may reach considerable size. Lymphatic and chylous cysts, caused by dilatation or bursting of the

lymphatics following occlusion of the thoracic duct or one of the larger lymphatic vessels, may also develop in the mesentery and retroperitoneal tissues.

The rare congenital blood cysts of the neck, which are the result of developmental disturbances in the anlage of the vessels and occur in positions ordinarily occupied by veins, the latter having failed to develop, are true cysts. The walls of these cysts correspond more or less closely to the walls of veins, and the contents consist of altered blood or of fresh blood, when the blind end of an imperfectly developed vein forms a sac. Blood cysts also develop along the course of the saphenous veins, when evaginations in varicose veins become constricted off from the parent stem.

True lymph or serous cysts are not easily recognized. The small cysts are simple dilatation of lymphatic vessels, while the larger cysts, if an endothelial lining is present, are to be regarded as cystic lymphangiomas. According to Odenius, however, cystic degeneration does occur in lymph nodes following lymph stasis. Nothing definite is known concerning the developmental history of the large chylous cysts which are occasionally found in the mesentery.

Multilocular cysts of lymph nodes, which are found most frequently in the groin, are called *lymphadenoceles.* These cysts are found most frequently in people in the tropics, and are the result of the stasis of lymph and the dilatation of the lymph sinuses following infection with the *filaria sanguinis hominis.*

(4) PARASITIC CYSTS

The echinococcus and cysticercus appear in the tissues as cysts which are encapsulated the same as foreign bodies.

(a) Tænia Echinococcus.—The tænia echinococcus is from 4 to 5 mm. long and possesses only four segments, of which the most posterior surpasses in length all the rest put together. A number of eggs are found in the last segment. The head is provided with sucking disks and a circle of hooklets.

Habitat.—The tænia echinococcus lives in the intestinal canal of dogs. It does not reach full development in man, but tends to the formation of large cysts which may seriously interfere with the function of the tissues and organs involved.

When mature the last segment becomes filled with ova which are discharged. These find their way into the human stomach in water or upon uncooked vegetables, such as water cress, which have been contaminated with the dog's excreta.

The process of digestion sets the embryos free, and they become at-

tached to the gastric and intestinal mucous membrane by the sucking disks and hooklets, and finally penetrate it to enter the lymphatic and blood vessels.

Transportation of Embryos by Blood and Lymph Stream.—They are then carried by the blood stream to the liver and with the chyle through the thoracic duct to the right heart and the lungs. The liver and the lungs act as filters, as frequently the capillaries are not large enough to permit of the passage of the large embryos of from 25 to 28 μ in diameter. After they pass through the vessel walls they develop to form large cysts, which may be unilocular or multilocular. According to the investigation of Mangold, Melniko-Raswedenkow, and Posselt, it depends upon the species of the worm whether unilocular or multilocular cysts develop.

Walls, Lining, and Contents of Cysts.—The embryo which has penetrated the tissues slowly becomes transformed into a small cyst which in the course of six months attains the size of a hazelnut. The wall of a hydatid cyst consists of a lamellar, stratified elastic membrane without cells (cuticula), the inner surface of which, the so-called parenchyma layer, contains muscle fibers, blood vessels, and the vesicular, germinal cells.

Echinococcus cysts are filled with a clear, watery fluid which contains no albumin, and therefore is not coagulated by heat. The fluid contains principally sodium chlorid, calcium oxalate, and a toxalbumin. The latter probably produces the toxic symptoms almost always accompanied by urticaria, which follow the rupture of a cyst. Scoleces and hooklets may be demonstrated microscopically in this fluid.

Daughter and Granddaughter Cysts.—When the embryo lodges in some viscus it changes into a cyst incapable of active motion. When the cyst has reached the size of a walnut approximately (sometimes earlier), there are formed from the parenchymatous layer small brood capsules, the delicate walls of which likewise consist of two layers, an inner cuticular layer and an outer parenchymatous layer. Upon these brood capsules the small heads or scoleces develop in very large numbers. According to Leuckart, they grow out of hollow sacs which bulge out from the external wall of the brood capsules. Brood capsules are formed in the same way as the primary cysts which develop from the six-hooked embryos. The daughter cysts which float free in the mother cyst may be very numerous, often as many as a thousand being found. They vary in size and sometimes contain granddaughter cysts.

If, after the development of brood capsules and scoleces, daughter cysts do not form, the echinococcus remains a simple cyst (acephalocyst), and is differentiated from the *echinococcus hydatidosus*, which is filled with daughter cysts.

Reaction of Surrounding Tissues—Rupture of Cysts.—The surrounding tissues form a connective-tissue capsule for the cysts, which in the course of years becomes quite thick. The capsule covering the part of the cyst which is exposed upon the surface of an organ is so thinned that it breaks easily. If the cyst ruptures, as frequently occurs, the daughter cysts and scoleces are scattered; for example, if a cyst ruptures into the peritoneal cavity the scoleces and daughter cysts become attached to the intestinal loops and numerous new cysts develop. If adhesions occur the cysts may rupture into the stomach, intestine, or trachea.

Infection of Cysts.—These cysts may become infected, undergoing suppurative or putrefactive changes. Rupture may then be followed by pleuritis or peritonitis, depending upon the situation of the cyst. Occasionally a cyst ruptures into the inferior vena cava, causing fatal embolism.

Death of Echinococcus with Spontaneous Cure.—Death of the echinococcus is a fairly frequent occurrence. It is observed when the cyst is small or after some disease of the host. The cyst then contracts to form a fatty or chalky mass, the nature of which can be recognized for a long time by hooklets and remnants of membrane.

Multilocular and Unilocular Echinococcus.—The *multilocular echinococcus* always develops small cysts which vary in size from that of a millet seed to that of a pea. These cysts are always present in large numbers. This echinococcus, which is found most frequently in the liver, lungs, and bones, appears as a firm tumor possessing upon section an alveolar structure, as it is composed of many small cavities surrounded by thick, compact, connective tissue. The contents of the cavities are gelatinous and transparent or of a semisolid, sometimes caseous, consistency. As a result of regressive changes in the thick connective tissues, developing secondary to the irritation of the cyst, large cystic cavities and extensive calcified areas composed of degenerated connective tissues and dead cysts may form. The tissues of cysts developing in the liver are often bile-stained. These cysts undergo an exogenous sprouting, invading the lymphatic spaces and vessels and producing a pressure atrophy of the viscera and tissues in which they are deposited. The cavities following regressive changes may rupture into the tissues adjacent to the organ primarily involved, or into a neighboring viscus which has gradually undergone atrophy as a result of pressure exerted by the cyst.

The two forms differ in their geographical distribution. The multilocular echinococcus is found most frequently in South Germany and Switzerland, where they also occur in cattle, more rarely in swine. The unilocular form, on the other hand, occurs most frequently in North

and East Germany, but also occurs over the whole of Europe, in South Australia, Algiers, Egypt, Cape Colony, and is especially common in Iceland.

The disease is extensively distributed among domestic animals, and among dogs in countries in which cattle are numerous. They have abundant opportunities for taking up the echinococcus, and therefore the danger of infecting the people is great.

Fig. 449.—Large Echinococcus Cyst in the Muscles of the Back (Echinococcus Hydatidosus).

Organs and Tissues Most Frequently Involved. — The echinococcus may develop in any part of the body. The liver and lungs are most frequently involved, however, as the embryos penetrate the blood or lymphatic vessels of the intestines and are deposited secondarily in the viscera above mentioned. Infection of the kidneys, spleen, peritoneum, muscles, bone, skin, mammary and thyroid glands occurs, but is much less common.

Prognosis.—The prognosis, like the symptoms, depends entirely upon the position of the cyst or cysts. Deep cysts, when they rupture or become infected, are much more dangerous than the superficial ones. Involvement of the vertebræ and pelvic bones is more fatal than that of other bones, as cysts of the vertebræ produce pressure upon the cord. Cysts of the pelvic bones frequently become infected and suppurate.

Symptoms.—Frequently the symptoms following the development of cysts in deep viscera (liver, lung, spleen, kidney) are indefinite. The symptoms first become definite when the cyst becomes large enough to exert pressure upon neighboring structures, suppurates or ruptures. Infection followed by suppuration is accompanied by severe pain, fever, rapid loss of strength, progressive inflammation of the organ involved,

localized or progressive suppuration in the peritoneal or pleural cavities, rupture into neighboring organs (stomach, intestines, urethra, bronchi), or through the skin. Rupture of the cyst, often produced by trauma, is followed by toxic symptoms (with urticaria), and the diffusion of brood capsules and scoleces from which new cysts may develop. Rupture is an especially dangerous accident when the peritoneum is involved. Death follows more frequently than spontaneous healing, rupture or suppuration of a cyst which is deeply situated.

Diagnosis.—The diagnosis belongs to the province of special surgery, as the symptoms are those which follow interference with the function of the organ involved. It should be mentioned that exploratory puncture should be omitted when the cyst is adjacent to the pleura or peritoneum, as the cyst may be ruptured and brood capsules and scoleces may be diffused.

The *external forms of echinococcus*—those of the subcutaneous tissues, muscles, intermuscular connective tissues, and superficial organs—appear as fluctuating growths which enlarge very slowly, often intermittently, and may exist for a number of years without causing symptoms. The surface of the swelling is smooth or nodular, when the brood capsules can be palpated. A cyst may become as large as a child's head or larger. The swelling is round or influenced by surrounding tissues, especially when it develops in the loose connective tissues between muscles or along large blood vessels. Even unilocular cysts are firmly attached by their connective-tissue capsules to the surrounding tissues, so that the overlying skin cannot be raised when the cysts are superficial, or the muscles separated from them when they are deep. The cysts are usually well defined, but can only be displaced with the surrounding tissues.

The external forms are found most frequently in the trunk and neck, the lumbar region, the abdominal wall, the axillary fossæ, and the inguinal regions. The spaces occupied by the vessels of the neck are most frequently involved. Of the different glands, the thyroid and the mammary are most frequently affected. Cysts of the extremity are found most frequently in the internal bicipital sulcus, in the region of the adductors of the thigh, and in the popliteal fossa. In the head they develop most frequently in the temporal and masseter muscles and the orbit.

The diagnosis of the external forms of echinococcus is usually made by exclusion and by the peculiarities of the parasitic cysts.

All fluctuating, circumscribed tumors, chronic abscesses, and cysts which occur in the area involved, such as cystic lymphangiomas, lipomas, dermoids, hygromas, cysts of the mammary and thyroid glands, ranula, and tuberculous abscesses, must be considered and excluded. The so-

called *hydatid fremitus*, which is elicited by tapping the cyst wall and is supposed to be characteristic, can be elicited only in the large unilocular cysts. The fremitus is probably due to the movement of the daughter cysts on one another. Aspiration of the cyst and examination of the fluid is naturally the most positive diagnostic method. This procedure, of course, is much less dangerous when the cyst is situated in the soft tissue than when it is situated in the pleural or peritoneal cavities.

A positive diagnosis is impossible when suppuration within the cyst or a pericystic phlegmon develops. The nature of the process in these cases is generally unsuspected until degenerating cysts and shreds of the cyst wall are revealed by incision.

Echinococcus of bone is not very common. Only one hundred and two cases have been reported up to the present time (Frangenheim).

The lesions, like embolic infections, are situated most frequently in the spongy ends of long bones. An old fracture or a part of a bone recently injured are most commonly attacked, the abnormal vascular relations in the callus and the lacerations of vessels produced by an injury providing favorable conditions for the deposition and growth of the parasite. The pelvic bones and vertebræ are next most frequently involved after the long bones. Isolated cases of involvement of the skull bones followed by rupture into the sphenoidal and frontal sinuses or into the cranial cavity, of the ribs, sternum, scapula, and phalanges have been observed.

The multilocular form develops in bones more frequently than the unilocular. In the beginning, cysts the size of a pinhead or pea develop in the spongy tissue. As a result of the atrophy of the bony trabeculæ and necrosis of the surrounding spongy tissue, large irregular cavities filled with yellow or white fluid, which contain sequestra of bone, bone sand, cysts, detritus, and cholesterin, are formed.

The cysts may be unobserved for a number of years and increase in size without producing any symptoms, or the latter may consist merely of mild migrating pains and a sense of weariness. The bone is destroyed gradually, and usually, even when the cyst extends to the periosteum, there is no periosteal bone formation to strengthen the part of the bone destroyed by the enlargement of the cysts. As a result the bone involved becomes very thin, and spontaneous fracture of the long bones or rupture of a puriform mass together with cysts into the soft tissues may be the first indication of the disease. The symptoms of echinococcus disease of the vertebræ are those of compression myelitis, caused by the destruction of the bodies of the vertebra involved and the development of extradural cysts. Flat bones, when involved,

become expanded, and "parchment crackling" or fluctuation can be elicited by palpation.

If an echinococcus cyst situated in an epiphysis ruptures into a joint, a large part of the latter is destroyed and a subluxation develops. The accumulation of material resembling that found in an abscess precedes rupture through the skin, which is followed by chronic fistula.

The *diagnosis* of echinococcus disease of bone is most difficult. The symptoms are so slight and indefinite and the disease so rare that a positive diagnosis is frequently not made until an operation is performed. Küster, for example, during an operation for pseudarthrosis which followed a second fracture of the humerus, found an echinococcus cyst. If a spontaneous fracture occurs, a myelogenous sarcoma, a tuberculous abscess, and gummata must be considered in determining the cause. If these can be excluded and there is no reactive proliferation of bone, it is probable that an echinococcus cyst is present. The findings elicited by palpation and an X-ray examination are very helpful. Abscesses and fistulæ suggest chronic suppurative or tuberculous osteomyelitis. Even bacteriological examination of the pus is not always positive, as the necrotic masses in a cyst frequently become infected with pyogenic bacteria. According to von Bergmann, the presence of numerous crystals of cholesterin in the aspirated fluid is the most important and positive finding in these cases.

Treatment.—In the treatment of echinococcus cysts an attempt should be made to remove completely the cyst together with the capsule, and to reëstablish normal conditions.

The unilocular cysts situated superficially are most easily removed. Frequently cysts which involve the peritoneum, mesentery, and omentum can be completely removed. When the cysts involve large viscera or large areas of the peritoneum or pleura, the hæmorrhage, following attempts at enucleation, is so severe that radical removal is impossible.

In these cases one must be content with incision and removal of the contents of the cyst after the cyst has been exposed and sutured to the edges of the wound. Drainage must then be continued for a number of months before healing is complete. The operation may be done in one sitting, that is, the cyst may be opened immediately after it is sutured into the wound, care being exercised to prevent the fluid from flowing back into the pleura or peritoneal cavities; or in two sittings, the incision of the cyst being postponed until firm adhesions have been established between the cyst wall and the edges of the wound.

As the period required for healing after incision as above described is prolonged, the operation has been somewhat modified. Bobrow and Garrè incise the capsule, after the surrounding tissues have been carefully protected, remove the contents of the cysts, and as much as pos-

sible of the membrane lining the inner surface. The parts of the capsule which can be easily separated are then cut away. The cyst is then closed by a double row of sutures and allowed to sink back into the abdomen. In order to protect against suppuration, and if recurrence is feared, the cyst may be sutured to the abdominal wall. If the cavity of the cyst and the transudate which forms remain sterile, the cyst gradually undergoes cicatricial contraction.

The removal of a multilocular echinococcus cyst from a viscus is very similar to the operations performed for malignant growths, and should be attempted only when the cyst is small (e. g., removal from the liver by cuneiform resection). The kidney, likewise the spleen when involved, should be removed. Incomplete operations, such as incision or removal of part of the cyst, are not successful.

Cysts of bone which are small may be easily exposed and satisfactorily removed by chiseling away the bone and curetting out the cysts. If the cysts are large and the bone has been extensively destroyed, resection should be considered; if the cyst has become infected and the general condition of the patient is poor, amputation.

Prophylaxis is of great importance. People who keep dogs should exercise great care. The segments of the echinococcus containing eggs are discharged in the fæces and the dog's nose may easily become infected. It is as dangerous to allow a dog to lick the hands and face as it is to use the plates from which a dog has eaten. It is important to keep dogs away from slaughter-houses, as they may become infected by eating material infected with echinococcus, and the disease may be spread in this way.

(b) Cysticercus Cellulosæ.—"The cyst provided with a tapeworm head is known as a '*measle*' or *cysticercus cellulosæ*. The scoleces, when fully developed, possess a circle of hooks, suckers, a water vascular system, and numerous calcareous bodies in their parenchyma. If they get into the human stomach the cyst is dissolved, and there develops, through formation of segments, a new chain of proglottides, a new Tænia solium."—Ziegler's "General Pathology," pp. 555–556.

The eggs of the tapeworm, derived from animals or man or from the patient himself, may be carried by infected drink or food or by unclean fingers into the mouth, reaching eventually the stomach, where the capsule surrounding them is digested by the gastric juice. The embryos are then carried by the lymphatic and blood vessels to different parts of the body, where they are deposited. They develop after some weeks (about nine) to form cysts (cysticercus) the size of a pea or cherry. These cysts remain viable for a number of years, and after death of the scolex cicatrize and become calcified. Occasionally cysts developing from the Tænia saginata are found in man.

Wherever the cysticercus is deposited a mild inflammation develops, which leads to a thickening of the connective tissues surrounding it. The cysticerci lying in loose tissue may migrate by their own movement.

The cysticercus develops most commonly in young people. A large number of cysts, from 100 to 1,000, may develop.

Muscles, subcutaneous tissues, the brain, and eye are most frequently involved. The liver and lung are more rarely attacked. Cysticercus disease of bone has been observed but twice.

The *symptoms* naturally depend upon the position of the cysts. Cysticercus disease of muscle and of the subcutaneous tissue is characterized by the development of round, firm nodules, which never become larger than a hazelnut. The muscles when involved become weak and there is general muscular pain. If the cysts rest upon nerves, paralysis, weakness, and neuralgia develop. If but a single cyst develops, it may be easily confused with a small tumor. Relatively frequently the brain and its membrane, more rarely the spinal cord, are involved in cysticercus disease, which occurs in these structures in a peculiar form, namely, in the form of cysts held together so as to resemble grapes (*cysticercus racemosus*). The cysts may produce no symptoms, or, depending upon their position, the symptoms of cortical epilepsy, or those of a tumor of the brain or spinal cord. They may cause death.

Cysticercus of the eye concerns the ophthalmologist. The cysts may develop in the orbit, the vitreous humor, beneath the retina, or conjunctiva, or in the anterior chamber, and cause destruction of the bulb.

The *treatment* consists of early removal of the cysts which are causing the trouble.

The frequency of the disease has decreased with the decrease in the frequency of tapeworm disease which has followed the inspection of meat, and the improvement in the methods of treating tapeworms.

LITERATURE.—*Aschoff.* Ergebnisse der allgemeinen Pathologie von Lubarsch und Ostertag. 2. Jahrgang, Wiesbaden, 1897.—*Askanazy.* Ein Fall von Cysticerkenbildung an der Gehirnbasis mit Arteritis obliterans cerebralis. Zieglers Beitr. z. path. Anat., Bd. 7, 1890, p. 83.—*v. Bergmann.* Ueber Echinokokken der langen Röhrenknochen. Arbeiten aus v. Bergmanns Klinik, Bd. 2, 1887, p. 1;—Handb. d. prakt. Chir., 3. Aufl., Bd. I, p. 26, Pneumatocele cranii.—*Chiari.* Ueber die Genese der Atheromzysten der Haut und des Unterhautzellgewebes. Zeitschr. f Heilk., Bd. 12, 1891, p. 189.—*Dannielsen.* Der Cysticercus cellulosæ im Muskel. Beitr. z. klin. Chir., Bd. 44, 1904, p. 238.—*Fischer.* Die Krankheiten der Lymphgefässe, Lymphdrüsen und Blutgefässe. Deutsche Chir., 1901, Lymphadenozele, p. 129.—*Gangolphe.* Maladies des os. Paris, 1894.—*Frangenheim.* Die chir. wichtigen Lokalisationen des Echinokokkus. Sammlung klin. Vorträge, 1906, Nos. 419–420;—Die chir. wichtigen Lokalisationen der tierischen Parasiten u. s. w. *Ibid.*, No. 424.—*Garrè.* Ueber neue Operationsmethoden des Echinokokkus. Beitr. z. klin. Chir., Bd. 24, 1899, p. 227.—*Gerulanos.* Das Vorkommen von multiplen Muskelechinokokken, nebst Bemerkungen über die Verbreitung der letzteren im Organismus. Deutsche Zeitschr. f. Chir., Bd. 48,

1898, p. 372.—*Gross.* Die Lymphangiektasie der Leiste u. a. I. Die Lehre der Adenolymphozele. Arch. f. klin. Chir., Bd. 76, 1905, p. 778.—*Hirschberg.* Cysticerken im Auge. Eulenburgs Realenzyklopädie, 1885.—*Fritz König.* Beitrag zur Anatomie der Dermoid- und Atheromzysten der Haut. Arch. f. Chir., Bd. 48, 1894, p. 184.—*Küttner.* Zysten der Speicheldrüsen. Handbuch d. prakt. Chir., 2. Aufl., Bd. 1, p. 666.—*Madelung.* Beitrag zur Lehre von der Echinokokkenkrankheit. Stuttgart, 1885.—*Mangold.* Ueber den multilokulären Echinokokkus und seine Taenie. Berl. klin. Wochenschr., 1892, p. 21.—*Marchand.* Zyste. In Eulenburgs Realenzyklopädie, Bd. 5, 1895, p. 256.—*Melnikow-Raswedenkow.* Studien über Alveolarechinokokkus. Zieglers Beitr. z. path. Anat., Bd. 4, Suppl., 1901.—*Mennicke.* Ueber 2 Fälle von Cysticercus racemosus. *Ibid.*, Bd. 21, 1897, p. 243.—*Odenius.* Ueber einfache zystische Degeneration d. Lymphdrüsen. Virchows Arch., Bd. 155, 1899, p. 465.—*Posselt.* Die Stellung des Alveolarechinokokkus. Münchn. med. Wochenschr., 1906, p. 537;—Die geographische Verbeitung des Blasenwurmleidens. Stuttgart, 1900.—*Riemann.* Ueber die Keimzerstreuung des Echinokkokus im Peritoneum. Beitr. z. klin. Chir., Bd. 24, 1899, p. 187.—*Speckert.* Ein Fall von Chyluszyste. Arch. f. klin. Chir., Bd. 75, 1905, p. 998. *H. Ström.* Ueber Pneumatocele cranii supramastoidea. Zentralbl. f. Chir., 1903, p. 1309.

APPENDICES

APPENDIX I

DIRECT TRANSFUSION OF BLOOD

The transference of whole or modified blood, by various methods, for numerous purposes, from an individual of the same or of an alien species to another has been practiced in many parts of the world for at least four centuries. The greater part of it was done crudely and empirically before the development of chemistry, physiology, pathology, and bacteriology, and before the period of good hospitals and surgical instruments. There were many accidents due to infection, clotting of the blood, the use of alien blood, and unfortunate selection of cases; so that with the advent of normal saline solution as a substitute for blood, transfusion of the latter was no longer practiced.

Crile in 1898 took up the work again, using the method introduced by Mosso. This method proved to be impractical, and the work lapsed until the work of Payr and Carrel and Guthrie gave better methods.

From clinical and experimental research Crile has come to the conclusion that the vascular systems of two patients may be united so that intima comes in contact with intima only. This may be accomplished by the special anastomosis tube devised by Crile, which is a modification of the magnesium tube introduced by Payr for arterial anastomosis, or by suture according to Carrel. The tube devised by Crile is made of German silver and is provided with two grooves upon its outer surface. The vessel is drawn through this tube and everted so that the intima is on the outer side. The vessel is then tied into the second groove, and the tube with intima on the outer side is then introduced into the vessel with which the anastomosis is to be made. Intima is thus brought in contact with intima, and there is no foreign body in the blood stream.

The blood may be transferred without clotting, the use of the radial artery of the donor and any superficial vein of the recipient yields the best results, the operation may be done painlessly, the blood lost by the donor is restored in from four to five days, and the amount transferred is under the immediate control of the operator. The rate of transference should be gauged carefully within the limits of physiologic safety.

Transfusion Cannula.—The first model for the transfusion cannula was suggested to Crile by Dr. Mixter in December, 1906. Dr. Mixter designed and constructed a splendid model made of two parts. Payr's magnesium tube gave some good suggestions as well. The cannula now in use answers the purpose splendidly, and was developed in its present form after more than twenty various models were made.

Management and Technic of Operation.—A suitable donor is usually readily obtained. We use both men and women. In cases in which no immediate emergency exists the most suitable subject is singled out from among the relatives and friends. He is approached tactfully, the most opportune time being just after he has left the bedside of the patient. The gravity of the patient's condition and the only means of relief are carefully detailed, the painlessness of the procedure to both donor and recipient being assured. Almost invariably a voluntary suggestion to serve as donor results. Indeed, frequently an entire family and friends have offered their services.

Our only difficulty, thus far, has arisen among ward patients who have a certain amount of distrust of surgeons and hospitals. Among these patients, however, I have experienced but one refusal, that being in the case of foreign parents of a child of nine, whose legs had been crushed, the argument being that the child was not worth saving. In two other instances the donors were hired. In these cases the commercial attitude was apparent and the donors were not as tractable as those who responded to the appeal of sentiment. A careful investigation as to the health of the donor, both as to whether or not it is advisable to remove blood and whether or not there is any disease which might be transmitted, is always made.

When there is time hæmolysis observations are obtained from the proposed donor and the recipient. This test requires about twenty-four hours. By making the hæmolysis test of the proposed donor and of the recipient various blood reactions may be obviated. Agglutination may, I think, with safety be disregarded.

The operating room should be equipped with two tables, preferably of the kind which permits of a change of posture from head-up to head-down. The patients are given pillows in order to be made as comfortable as possible, and are so arranged that the left arm of each may be used. The donor should be placed on the table so that, if necessary, the Trendelenburg position may be utilized. The recipient, if both postures are not available, should be arranged so that the reverse Trendelenburg may be given. This permits the better management of a possible dilatation of the heart of the recipient and of a cerebral anæmia of the donor. I have found that it is a great aid to have a trained operative staff, so that the many details may be performed without delay and

without speaking. Two small movable tables, the height of the operating tables, are most convenient for supporting the arms and the instruments during the dissection. One of these tables will support both arms during the process of making the anastomosis and during the remainder of the transfusion. On either side of this table and between the two operating tables a stool is placed, which provides a comfortable and a steady position for the operator and his first assistant *vis-à-vis*. From the beginning until the end not an unnecessary word is spoken. Both the donor and the recipient, unless contra-indicated, are given a preliminary hypodermic of ¼ grain of morphin twenty to thirty minutes prior to their entrance to the operating room. The patients are assured that they will experience no pain, save the first needle prick. In order that they may not obtain a glimpse of the operating room or of their environment, both patients are told that, owing to the bright light, wet towels will cover their eyes, thus preventing a possible headache. They are warned of the first needle prick, and are told that cocain will now be administered, that it will require twenty or thirty minutes to take effect, and that in the meantime it will be necessary to massage, to prick, and to pull the tissues, but that the procedure is painless. One nurse is detailed to relieve the monotony of waiting by substituting fresh towels, bathing the brow, administering water if desired, and giving helpful attention.

Local anæsthesia is maintained by infiltration of one tenth per cent solution of cocain with a few drops of adrenalin, first in the skin proper, and then in the neighborhood of the vessels, after which firm pressure for thorough dissemination is applied. When carefully performed there is absolutely no pain in any part of the procedure until the suture of the skin at the close of the transfusion, at which time the effect of the cocain has disappeared. In the dissection I have found it an advantage to use minute instruments, selecting from among the armamentarium of oculists and watchmakers. Mosquito forceps are used to catch every vessel that sheds even a drop of blood, keeping the field not only clean but translucent. The donor's radial artery is isolated a distance of about 3 cm. At the point of election there are a number of small branches which should be carefully isolated and tied, otherwise an obscuring hæmorrhage may occur. The small nerve branches and the venæ comites are pushed aside. The artery is then tied at its distal end, and at the proximal a screw clamp gently closes its lumen. The artery is then divided with a sharp scissors, the adventitia is drawn well over its end and snipped off closely. This leaves a clean open end of the vessel, but the manipulation and exposure to the air causes such sharp contractions that for a time its lumen is obliterated. This is easily overcome by inserting into the lumen a mosquito forceps, covered

with vaseline, then gently opening the blades. This overstretching of the artery's lumen prevents recontraction. Any superficial vein that seems neither too large nor too small is likewise exposed, isolated, ligated at its distal end, closed by a screw clamp at its proximal part, divided near the ligature with a sharp scissors, and its adventitia drawn well out over the end and snipped off closely, thus leaving a free manipulable end. The tables of the donor and of the recipient are approximated with their heads in opposite directions, so that the vessels may be approximated more readily and the stream may be transferred in nearly a straight line.

The vessels are now compared with the various sizes of the transfusion cannulæ and a suitable one selected. Then with mosquito forceps the handle of the cannula is grasped and the cannula dipped in sterilized vaseline or oil. The vein is next pushed through the lumen. With oculist's small, self-locking forceps or mosquito hemostats the margin of the vein is grasped, turning it back as a cuff over the outside of the cannula, and a fine ligature of linen tied firmly around the cuff in the second groove, the ends of the ligature being cut off. With one hand the cannula is steadied by means of the hemostat, and with small, locking thumb forceps or mosquito hemostats the assistant and operator grasp the end of the artery at three equidistant points and draw it over the venous cuff and cannula, tying it snugly with a small linen ligature in the first groove, thus completing the anastomosis. The screw clamp is then removed first from the vein, then from the artery, and the flow tested. At first, owing to the great contraction of the artery, but little blood flows across, but by liberal application of warm salt solution the vessel soon dilates and the stream grows larger, reaching its maximum in about ten minutes. It is most important not to bruise the vessels or to break the intima. In every instance in the 51 clinical cases the technic was entirely successful.

CONCLUSIONS

The principal danger of transfusion, now that the technic is perfected, is hæmolysis. This apparently occurs only in disease. The dangers of hæmolysis may be prevented by determining before the operation is undertaken whether the blood of the donor is hæmolytic for that of the recipient.

Sufficient facts have been determined by laboratory experiments and clinical observations to justify the following conclusions: Transfusion, when properly safeguarded, may be safely done. In pernicious anæmia, toxæmia, certain drug poisonings, leukæmia, acute hyperthyroidism, and uræmia, it has been of no value. In tuberculosis, carcinoma, and chronic

infections it is of doubtful or at best little value. In human sarcoma there is some evidence of value, though not yet proved. In pathological hæmorrhage it is of marked value. In suitable cases it seems to be almost a specific in the prevention and treatment of shock. In acute hæmorrhage in animals it is specific; in human beings it has proved most valuable.

APPENDIX II

OPSONINS, PHAGOCYTOSIS, AND THE THERAPEUTIC INOCULATION OF DEAD BACTERIA

THE discovery of opsonins by Wright and Douglas and the introduction of the inoculation of dead bacteria for therapeutic purposes mark an important epoch in the studies of immunity. It cannot be said at present what the ultimate results of vaccine therapy will be, but the outlook is so promising that a brief consideration of the nature of opsonins and the value of the inoculation of dead bacteria is presented here.

The discovery by Wright and Douglas that the serum from normal and immune blood contains substances, called by them opsonins,[1] which have the power to render bacteria susceptible to phagocytosis, has reawakened interest in Metschnikoff's theory of phagocytic immunity. It has been shown conclusively that the phagocytosis of most bacteria by leucocytes depends upon the action of serum upon the bacteria, which in some way changes them so that they are freely taken up by polymorphonuclear leucocytes. Bacteria suspended in salt solution resist phagocytosis by washed leucocytes, while bacteria previously treated by serum and then washed, i. e., freed from serum, are taken up readily by washed leucocytes. Bacteria so treated are said to be sensitized or opsonified. The character of this change is wholly unknown. There is no recognizable alteration in form, staining reaction, or function of the bacteria. Many bacteria grow freely in sera which contain opsonin.

Normal opsonins are largely destroyed by heating the serum to 60° C. for thirty minutes, some being more resistant than others. Immune opsonins, those produced as the result of infection or experimental inoculation of bacteria, are more resistant. This difference is attributed by most authors to their greater concentration. This view is supported in a measure by the fact that diluted immune serum frequently shows a high opsonic value; when normal serum controls show none. The

[1] From the Latin *obsono* or *opsono*, "I cater to," "I prepare food for."

whole question whether normal and immune opsonins are identical or not is still unsettled.

The *opsonic index* represents the relative amount of an opsonin in the serum of an individual as compared with a normal standard in that case. The opsonic index with reference to a given bacterium is obtained by dividing the average number of bacteria taken up per leucocyte under the influence of the patient's serum by the average number taken up per leucocyte under the influence of the standard normal serum under conditions which are comparable. The difficulties and the numerous sources of error in the determination of the opsonic index are claimed by some to be so great as to render the results unreliable. The uniformity of results of investigators under similar conditions, their agreement with what one would expect upon clinical and other grounds, would seem to indicate that in competent hands the method is of distinct value. But that it cannot be relied upon as an index of the antibacterial power of the individual under all circumstances is certain, because opsonin, it must be remembered, constitutes only one of the antibodies produced in the reactions of immunity.

It is too early to speak definitely upon the *diagnostic value* of the opsonic index. The conclusions of different investigators vary within wide limits, some attributing to it much, others little or no diagnostic value. From the evidence at hand it is certain that it can never be used as a routine measure for the identification of infections, because the index may be high or low in a given instance, usually depending upon whether the patient is on the improve or not. But that it may be of distinct value in certain conditions, just as the agglutinization of typhoid bacilli is valuable in the diagnosis of typhoid fever, is quite likely.

The evidence that *inoculation of suitable bacteria* in proper amounts usually causes an increase in the opsonic power of the serum with respect to the organism inoculated is convincing. This is true in the normal individual or animal as well as in chronic infections due to the corresponding micro-organism. Denys found that rabbit leucocytes in normal serum ingested virulent streptococci, but not those made virulent by repeated passage through animals. In the serum of the immunized rabbits and horses the leucocytes showed decided phagocytic power over virulent streptococci. Bordet, Besredka, and v. Lingelsheim all noted the greatly increased phagocytosis of streptococci in the presence of immune serum both in vivo and in vitro. Metschnikoff believed this increased phagocytosis to be due to a stimulation of the leucocytes, and designated the substances assumed to stimulate the phagocytes as "stimulins." In the light of the opsonic theory much of what was thought to be due to the stimulation of the leucocytes is in reality the result of opsonification. Neufeld and Rimpau have shown that leuco-

cytes digested in antistreptococcic serum and then suspended in normal serum failed to take up virulent streptococci. But digestion of virulent streptococci in antistreptococcic serum, then washed in normal salt solution and mixed with leucocytes, resulted in marked phagocytosis, thus showing that in this instance immune serum may so change virulent streptococci that leucocytes ingest them. Rosenow has not been able to render virulent pneumococci susceptible to phagocytosis by normal or immune serum, including some of the so-called antipneumococcic sera. The demonstration that opsonins render various bacteria susceptible to phagocytosis does not prove fully that they are of any importance in combating infections. It must be shown that phagocytosis is the essential factor in the destruction of certain bacteria by the blood. Hektoen has shown that in all probability the relative immunity of a dog to anthrax is due to phagocytosis. Virulent anthrax bacilli grow freely in dog serum, but are destroyed in defibrinated blood as the result of phagocytosis. Denys showed that in mixtures of normal rabbit leucocytes and normal rabbit serum there was little or no destruction of virulent streptococci. Whereas, when immune serum was substituted, prompt phagocytosis and destruction of streptococci took place. The serum of normal persons and patients with streptococcus infections is a good culture medium for streptococci. Ruediger has shown that normal defibrinated blood, as well as the blood from patients with acute infections, have a streptococcidal effect which is roughly proportionate to the number of leucocytes present. He shows, too, that the destruction of streptococci requires the presence of opsonins. Rosenow has made analogous observations with respect to the pneumococcus.

In practically all experimental work in this field the phagocytic value of leucocytes is considered the same. Rosenow and Potter, on the other hand, have shown that there may be a distinct difference. The former observer found a greater phagocytic activity of leucocytes obtained from cases of lobar pneumonia, endocarditis, and other acute infections associated with leucocytosis. The difference was so great in a number of instances that pneumococci of a grade of virulence which resisted normal leucocytes were taken up by the leucocytes engaged in the infection under identical conditions. Important as these observations are, it must be remembered that they are made with bacteria grown upon artificial media, and hence under conditions very different from those in the tissue fluids or blood. The changes which the infecting bacteria may acquire to protect themselves against the action of animal antibodies, according to the theory of Welch, must be distinctly borne in mind in this connection. The prompt and pronounced phagocytosis of different bacteria in the peritoneal cavity in the presence of specific immune serum would seem to indicate that opsonins

play the same part in vivo as in vitro. That phagocytosis helps the body to rid itself of some bacteria is certain, but whether opsonification and phagocytosis play the primary rôle or a secondary rôle is still doubtful. However this may be, we have in opsonins a new form of antibody that must be considered, especially in the explanation of immunity to those infections caused by bacteria whose destruction is not accomplished by free lysins (streptococci, pneumococci, etc.).

The *injection* of *bacterial products* for curative purposes originated with Koch when he introduced tuberculin as a remedy for tuberculosis. Petruschky and Richardson tried to hasten the reactions in the healing process of typhoid fever by injecting products of the typhoid bacillus. Wright and Douglas first noted the rise in the opsonic power of the serum by injection of dead bacteria in chronic staphylococcus infections. They also showed that the opsonic power for tubercle bacilli in tuberculosis greatly increases in response to the injection of tuberculin. Upon these and other observations Wright places the method of treating infections by the injection of the corresponding dead bacteria or bacterial products upon a scientific basis. The opsonic index is used as a guide for the time that the injection is to be made and the amount to be injected.

Hektoen summarizes as follows the considerations for the therapeutic inoculation of dead bacteria:

First, the power of the injected bacterial substances to stimulate the formation of opsonins and other specific antibodies.

Second, the belief that increased formation of such substances may hasten healing of the corresponding infection.

Third, the apparent inability of the body under certain conditions of natural infection to produce such substances in sufficient quantities without special stimulation.

The essential prerequisites for therapeutic inoculations are:

First, correct etiologic diagnosis.

Second, sterilized, pure cultures of the bacterium causing the infection in each disease or sterile products of such bacteria; and,

Third, the injection of proper doses at proper intervals so as not to unnecessarily lower the antibacterial power or cause other unfavorable disturbances.

Experimental inoculation as well as auto-inoculation have a favorable effect upon the course of some chronic infections, provided, as indicated above, they do not overstimulate the reactive powers of the organism. The essential feature of the Wright method is the use of doses of dead bacteria just sufficient to raise the opsonic index above normal and keep it there.

In the tuberculin treatment of localized tuberculosis, Wright and

his adherents do not aim at a tuberculin immunization, but content themselves by keeping the opsonic index above normal. The dose of tuberculin remains very small throughout. The method used by Trudeau and others consists in giving progressively increasing doses of tuberculin, but just small enough to avoid a clinical reaction. Tuberculous patients may be made insusceptible to ten thousand times the amount of tuberculin which would cause an initial reaction. In this method no attention is paid to the opsonic index. The coincident improvement of the patient's condition so treated goes to show that the progressively increasing doses of tuberculin without reference to opsonin need not be harmful. The former method has been of apparent value in the treatment of chronic localized tuberculosis, excepting the pulmonary form. Its value in pulmonary tuberculosis is not so definitely established. The latter method, which has been used for a much longer period, chiefly in pulmonary tuberculosis, is believed by the best authorities to have a beneficial effect in many cases. That we have in tuberculin a powerful remedy for the treatment of certain chronic cases can scarcely be questioned. But that we have still much to learn of its therapeutic indications is equally certain. The reports of its good effects are not sufficient to warrant the giving up of the well-established surgical methods, but surely the surgeon who does not use it in conjunction with other methods, or give it a trial where other methods fail, is open to criticism.

It is impossible to estimate at this time how much reliance is to be placed upon the therapeutic inoculation of dead bacteria, because as yet chiefly isolated cases have been reported. But since the diseases in which such good results have been reported recover spontaneously, more extensive statistics and greater experience are needed before any satisfactory conclusions concerning the value of the therapeutic inoculation of dead bacteria can be made.

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